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ANNUAL

OF THE

UNIVERSAL MEDICAL SCIENCES

A YEARLY REPORT OF THE PROGRESS OF THE GENERAL
SANITARY SCIENCES THROUGHOUT THE WORLD.

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ASSISTED BY

OVER TWO HUNDRED CORRESPONDING EDITORS, COLLABORATORS,
AND CORRESPONDENTS.

Illustrated with Chromo-Lithographs, Engravings and Maps.

VOLUME I.

1888.

PHILADELPHIA AND LONDON:
F. A. DAVIS, PUBLISHER.

THE OCEANIC PUB. CO., Sydney, N. S. Wales. ....AGENCIES............ J. A. AIKEN, Cape Town, Cape Colony.

Entered according to Act of Congress, in the year 1888, by
F. A. DAVIS,
In the Office of the Librarian of Congress at Washington, D.C.

Philadelphia:
The Medical Bulletin Printing House,
No. 1231 Filbert Street.

27 Je '11 A. P. L.

PREFACE.

THE object of the ANNUAL OF THE UNIVERSAL MEDICAL SCIENCES is to collate the progressive features of medical literature at large, and clinical data from countries in which no literature exists, and to present the whole once a year in a continued form, prepared by writers of known ability. As such it is expected to become a helpmate to the practitioner in his efforts to relieve suffering, and to assist the investigator by correlating facts, thus enabling him to better compare. How far these ends have been attained the editor leaves to the decision of the profession,—to which he humbly dedicates the work, with the assurance that he has striven conscientiously to do his duty.

To the gentlemen forming the associate staff the successful issue of the ANNUAL is principally due, the support they so promptly granted it when the plan was first presented to them, and their subsequent interest during the year—not to speak of their valuable contributions—having proven most potent elements in the consummation of the work. As to the corresponding staff, the columns of the ANNUAL will show how useful their contributions have been in the preparation of the general articles. To them, also, the editor wishes to express his gratitude, and the hope that they will appreciate as much as he does the value of their assistance. He much regrets that very interesting papers by Professor Rossbach, of Jena, Professor Sänger, of Leipsic, Professor Maklakoff, of Moscow, Dr. Arthur Neve, of Bombay, Dr. Thompson, of Petchaburee, Siam, Dr. John

Reid, of Melbourne, Dr. F. Rincon, of Caracas, Venezuela, and several others, came too late to be of service for this issue. The majority of these contributions, however, fortunately come within the scope of the next series.

For any shortcomings the editor must appeal to the indulgence of his readers. The fact that the literature of the year was furnished by him to each associate editor, —making it necessary to classify and distribute not only the reports of the corresponding staff, which comprised a vast quantity of manuscript, but upwards of fifty thousand journal articles,—on the one hand, and the laudable desire of the publisher to deliver the work to its subscribers promptly on the date promised, on the other, made the editor's task arduous in the extreme. To render prompt delivery possible the entire series of articles, requested by the first of February, had to be prepared for the press, read in proof four times, printed and made ready for the binder within twelve weeks. The fact that this was accomplished reflects as much credit upon the associate staff as it does upon the publishing department. It involved, however, a few oversights, among which is the much regretted omission of the names of the following journals under cuts reproduced from their columns:—

Volume III.—*Canadian Practitioner*, p. 15; *Archives of Ophthalmology*, p. 19; *Ophthalmic Review*, p. 28; *New York Medical Journal*, p. 300; *Wiener Medizinische Wochenschrift*, p. 331. Volume IV.—*Revue Médicale de la Suisse Romande*, p. 8; *British Gynæcological Journal*, p. 9; and the *American Practitioner and News*, pp. 406 and 407.

In this connection the editor wishes to express his grateful acknowledgment of the courtesy extended to the ANNUAL by the journals and publishing firms generally, including,—

Messrs. D. Appleton & Co., of New York, Lea Brothers & Co., P. Blakiston, Son & Co., and the S. S. White Dental Co., of Philadelphia,—who kindly permitted the reproduction of cuts.

In the reference lists, it was the editor's intention to mention the name of the journal quoted in each instance; but this was found to take up so much space that after a few articles had been printed it became necessary to modify the plan, and to name each journal but once in the list,—the number representing the periodical being repeated in the text as many times as it was quoted. Want of space also prevented the mention of the date, volume, etc., of the original articles quoted. Arrangements will be made, however, by which this defect will be remedied in the next series.

The order of the articles will be found somewhat unusual in a few instances. This is due to the irregularity with which the contributions were received, some of those originally placed at the beginning of a volume having been the last to arrive, thereby rendering it necessary to modify the sequence of the articles at least thirty times. It should not be inferred, however, that the last articles, as now presented, were the last to reach the editorial department, some of these having been drawn down by neighbors whose subjects could not be separated from theirs.

The general style of five of the articles will be found to vary somewhat from the general plan of the work. This is due to the fact that it was deemed advisable in the first issue to treat a few subjects not usually found in text-books *in extenso*, in order to enable the subscriber to possess a complete review of the subject.

One of the articles on dentistry could not be published, its editors having been unable, through unavoidable circumstances, to prepare it in time. It will appear in the forthcoming number of the SATELLITE, with twenty additional pages.

The index, as presented, was devised with a view to better carry out the plan of the work as regards its practical usefulness. Divided into three columns, each has its own separate object. The first is the general index; the second is a ready reference list in which the practitioner can see at a glance, and in time of need, what therapeutical suggestions of practical value may have been made during the year in the treatment of any particular disease. In the third column is a general list of all the authors quoted throughout the entire work, so grouped as to enable the investigator to find at once all allusions made to any subject that he may be studying. How admirably Dr. Witherstine and his collaborators have carried out this plan, rendered exceedingly difficult by the short time that could be given them, will doubtless be appreciated by the readers of the ANNUAL.

The transfer of the table of contents to the cover will doubtless prove more convenient in that situation. In addition, page headings have been inserted throughout the work, to facilitate the search for any particular subject.

On account of ill-health, the ANNUAL lost during the year the valuable assistance of Drs. F. N. Otis, of New York, and J. B. Roberts, of Philadelphia, much to the regret of the editor. Drs. F. R. Sturgis and Henry M. Lyman, who kindly undertook their articles, will be found to have prepared them in a manner quite in accord with their high standing as writers.

It becomes necessary to record the early and sudden demise of one of the associate editors of the ANNUAL, Professor N. Archer Randolph, of the University of Pennsylvania. The high professional attainments and personal qualities that distinguished him made his loss a great one to science and to his many friends. The department of hygiene, which he had undertaken, was ably edited by Dr. John B. Hamilton, of Washington.

The editor does not wish to close without expressing his great appreciation of the assistance rendered him by his publishers. He desires to state also that had they confined themselves to their first estimate as to cost,—although this would have enabled them to satisfactorily fulfill their contract with the subscribers,—much could have been saved; but their anxiety to enhance as much as possible the value of the work to the profession led them not to hesitate in carrying out any suggestion or granting any request of the editor, at whatever cost.

To Messrs. Ferguson Bros. & Co., printers, Burk & McFetridge, lithographers, The MacKellar, Smiths & Jordan Company, electrotypers, Fickinger & Stowell, engravers, and Oldach & Co., bookbinders, much credit is due for their prompt and excellent work

THE EDITOR.

Philadelphia, April 21st, 1888.

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DISEASES OF THE BRAIN AND SPINAL CORD.

By E. C. SEGUIN, M.D.,

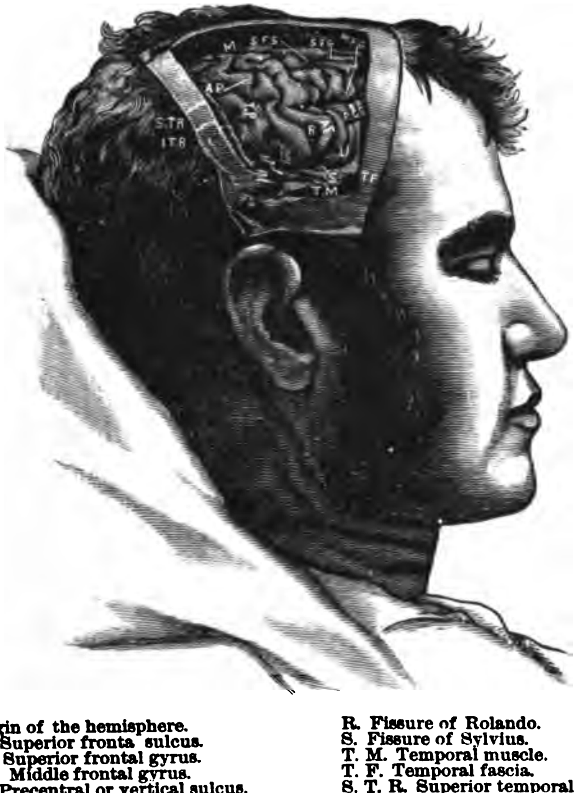
NEW YORK.

CEREBRAL AND SPINAL LOCALIZATIONS.

No important discovery has been added to our knowledge of these subjects during the past year, but several instructive discussions have taken place in scientific societies on them, and quite a series of new facts, experimental and autopsical, have been recorded. The results have tended to strengthen the doctrine of cerebral cortical localization in man, and the more exact application of this doctrine deductively to medical diagnosis and surgical treatment. Whatever disputes may still go on between physiologists as to some of the centres in animals, their precise location, their *modus agendi*, etc., we clinicians and practitioners have received only confirmatory evidence of the actual tangible existence of such centres in the human brain, and localization diagnoses are made with greater and greater positiveness.

Cerebral Localization.—Of the formal essays on the subject that of Horsley[1] is the most elaborate, if not the most valuable. Its title, "A Note on the Means of Topographical Diagnosis of Focal Diseases affecting the So-called Motor Region of the Cerebral Cortex," indicates its scope. Horsley prefers the method of Professor Thane and himself for the determination of cranio-cerebral anatomy. He finds the upper end of the Rolandic fissure by taking "the length of the middle line of the head from the root of the nose—*i. e.*, the glabella—to the occipital protuberance or inion, and then by halving the result to obtain the centre of the middle line of the head. The upper end of the fissure of Rolando will be found in adults to be situated half an inch behind this centre-point of the head." The fissure of Rolando extends downward and forward at an angle of 67°, not, of course in a perfectly straight line. In the determination of other sulci and gyri, sutures and prominences are relied upon as landmarks. "The sutures occupy, of course, the foremost position among the *points d'appuis* of topography." We cannot fully agree with Horsley on this matter. To our mind, and in our hands, the Broca projections from the alveolo-condyloid plane are more practical, *i.e.*, more applicable to the living patient with scalp uncut. It is approximately exact, and Horsley himself makes no special criticism of it. But, according to race, sex, size, and shape of skull, etc., every method of cranio-cerebral topography fails to be geometrically exact; yet as we have to deal with gyri and centres which measure from 5 to 10 mm. across, minute errors can be set aside, especially if a large exploratory opening is to be made in the skull. Even without measurement one accustomed to the study of crania and brains should be able to trace out the fissures of Rolando and of Sylvius with his finger, with the patient's head accurately placed in Broca's position. The rest of the paper is made up of quotations with illustrations from series of experiments by Dr. Beevor and the author upon the cortex of monkeys (electrical excitation experiments), and reference to four cases of human cerebral lesion operated upon by Horsley. These statements are simply corroborative of already acquired knowledge, experimental and pathological. In this paper, as in others, Mr. Horsley appears to assume that our progress in cerebral localization has been mainly dependent upon experimentation. Here again we must differ from him. Clinical observation and pathological data come first (Broca for speech-centre, Hughlings-Jackson for a hand-centre and general doctrine), the animal experiments with detailed proofs by Hitzig, Ferrier, and others long after; and the solid facts upon which we make our daily localization diagnoses have been patiently accumulated by pathologists, and would stand to-day supporting the doctrine of cerebral localization if not one animal's brain had been touched. Besides, in the case of the visual half-centre, human pathological facts have overthrown the results of experimentation (Ferrier's angular-gyrus centre) and have made us, for practical purposes, indifferent to the contradictory results of Munk and Goltz. It is safe to assert that every one of the so-called "centres" in the human brain have been determined empirically by post-mortem proofs, independently of experimental data. What animal experiments would have led us, for example, to locate the half-centre for ordinary vision in the cuneus, the centre for the leg in the paracentral lobule, and that for audited language in the left first temporal gyrus? In this department of pathology medical science has been strictly inductive and sufficient unto itself, though receiving confirmatory evidence from the physiologist. The first (speech) and the last (visual) centres have been discovered by clinical and pathological studies.

Horsley's figure is reproduced partly to illustrate his topographical method, and partly because of its value for surgical purposes. The figures illustrating Broca's method of cranio-cerebral topography will be found on p. 42 of vol. ii. of Gross' "System of Surgery," Phila., 1882, and Pepper's "System of Medicine," vol. v., p. 94.

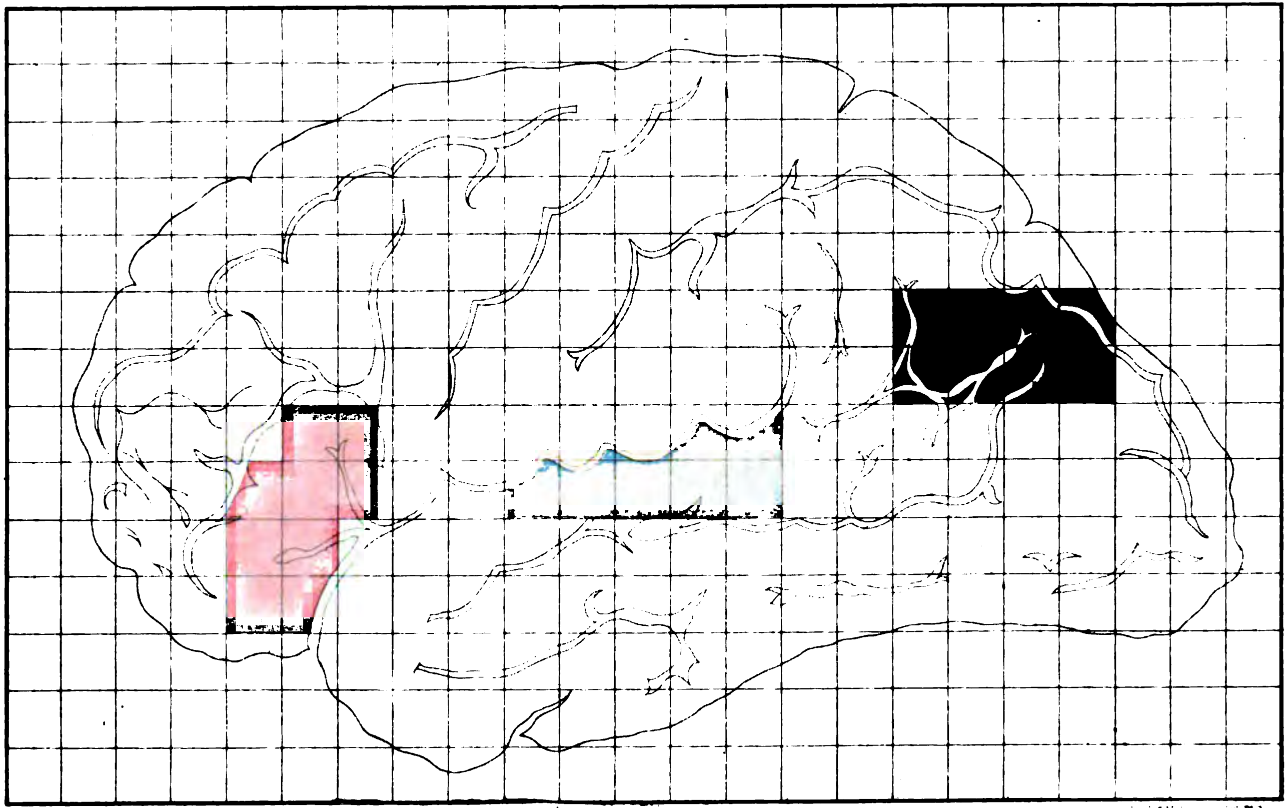


M. Margin of the hemisphere.
S. F. S. Superior frontal sulcus.
S. F. G. Superior frontal gyrus.
M. F. G. Middle frontal gyrus.
P. C. S. Precentral or vertical sulcus.
A. F. Ascending frontal gyrus.
A. P. Ascending parietal gyrus.
I. P. Intraparietal sulcus.
R. Fissure of Rolando.
S. Fissure of Sylvius.
T. M. Temporal muscle.
T. F. Temporal fascia.
S. T. R. Superior temporal ridge.
I. T. R. Inferior temporal ridge.
U. Subordinate sulcus in ascending frontal gyrus which unites the phonotory centre.

(*Am. Jour. Med. Sciences.*)

At the meeting of the Sixth Congress for Internal Medicine held at Wiesbaden, two very important referees' papers on cerebral localization were presented. The first, by Nothnagel,[2] relates to the localization of lesions in the visual centre and in the various motor centres. The author considers it as demonstrated that in the human brain there is localization of function in various parts of the cortex. As regards hemianopsia, he admits that its lesion involves one cuneus, but would add to this as part of the centre gyrus O^1, which we are not prepared to contest. He explains the hemianopsia caused by lesion of the angular gyrus or the inferior parietal lobule by injury to the subjacent fasciculus opticus, without stating that this explanation was first advanced by the writer of this review in 1886.[3] The remaining occipital convolutions constitute an additional centre for visual residua, and lesion of them causes psychic or soul blindness, as contradistinguished from coarse objective vision. The precise localization or limitation of this area for psychic vision is as yet undetermined. He suggests that as the occipital gyri exhibit frequent variations in form and arrangement, we may look for somewhat variable localization of functions. The paragraphs on motor localization contain several interesting propositions. In the first place Nothnagel declares that he has never observed loss of muscular sense in cases of lesion limited to the motor area, though a degree of tactile anæsthesia is usually present. In such cases the patient, though the arm or leg is completely paralyzed, yet remembers and is conscious of the nature and direction of movements he would wish to make. In the second place, Nothnagel finds, with a few authors preceding him (myself among them[4]), that lesions of the inferior parietal lobule produce loss of muscular sense with little or no paralysis or analgesia. From these facts he draws the important conclusions, which seem to us warranted by the facts now in our possession, that the parietal lobe in its inferior part contains a centre for the registration of motor residua, and that the centres in the motor zone are really motor centres.

The second paper, by Naunyn,[5] is an excellent example of the inductive geometric method, somewhat after Exner, relative to cases of aphasia from cortical lesions. He analyzes 71 cases of aphasia, classified as motor, sensory (acoustic), and undefined aphasia. He then locates the numbers representing the lesions of these cases upon a diagram of the cerebral hemisphere subdivided



Maximum foci of disease in 71 cases of Aphasia. (Naunyn)

🟥. Cortical area for motor aphasia (Broca.)

🟦 Cortical area for aphasia with word-deafness (Wernicke.)

⬛ Cortical area for aphasia with alexia or word-blindness

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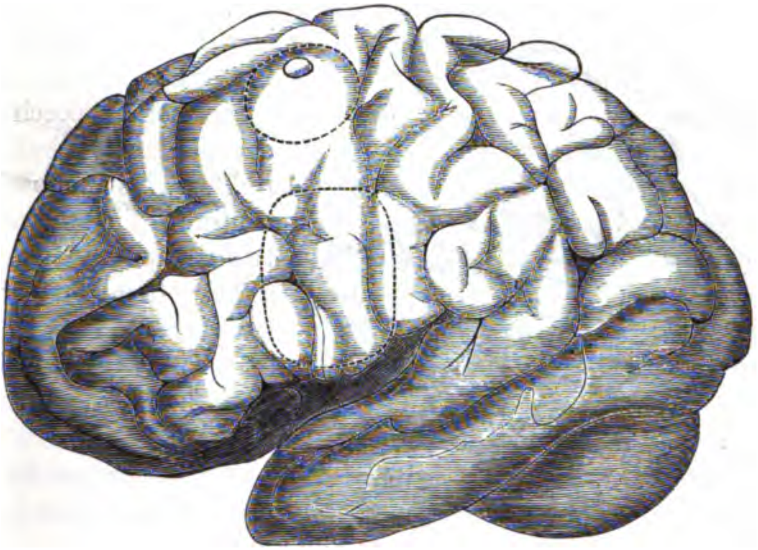
into small squares: the accumulation of figures in certain areas of the diagram indicates the centres for various special functions. As a result, he finds that the lesion of motor aphasia (true aphasia) is in the basal part (foot) of the left third frontal gyrus, or Broca's centre; that the lesion of sensory aphasia with word-deafness is in the upper extremity of the first temporal gyrus (Wernicke's centre); and that in all probability the lesion in cases of word-blindness (alexia) is in the parieto-occipital area, involving the posterior part of the inferior parietal lobule and the first occipital gyrus. The plates illustrating the general results are herewith reproduced. Naunyn supports the now classical doctrine that all aphasia-producing lesions are in the left hemisphere, except in the case of truly left-handed persons, when they are in the right hemisphere. In this, as in almost all recent contributions, Broca's proposition, formulated in 1861, long before any physiological demonstration of "cortical centres," is sustained,—a striking evidence of the superiority of the clinico-pathological method of study. It should be added that Naunyn guards his conclusions with the statement that he does not think that the above-mentioned centres or areas have definite limits, and that there may be variations in different human brains as regards fasciculation of white substance and superficial arrangement of corresponding cortical areas; all of which is, we may say, a generally admitted qualification to the doctrine of cortical localizations.

In striking contrast to these two valuable papers, produced by strict adhesion to the inductive method, is the report of the discussion on the localization of the muscular sense in the Neurological Society of London, December 16, 1886.[6] Although frequent reference is made to physiological experiments (mostly English), the whole form of the discussion is unscientific and abstract; entire series of known facts are left out of consideration, which, while they do not fully settle the question under debate, yet to a certain extent simplify it and point out the way for further post-mortem study. We refer more especially to the fact that in cases of destructive lesions of the motor gyri or of parts of them, the muscular sense is not lost; to the fact that when the lesion affecting the motor zone extends caudad, so as to involve the parietal lobules (the inferior particularly), the muscular sense *is* lost; to the few but most valuable facts that when a lesion destroys the inferior parietal lobule alone, or not much besides, there is loss of muscular sense in the opposite limbs, with paresis or without. The impairment of the muscular sense or the loss of remembrance of residua of muscular movement, is shown by coarse incoördination, not true ataxia, and by the testimony of the patient in response to tests by passive movements, weights, and attempted movements without the aid of vision. Other series of facts bearing upon the question whether the gray substance of the motor area is really motor, are barely referred to. We refer to the facts that descending degeneration follows the removal of the motor cortex, and that faradization of the fasciculi underlying and connecting with the motor cortex produces movements in the limbs of the opposite side. It seems to us that a comparison of this discussion with the papers of Nothnagel and Naunyn, with the classic collection of cases by Charcot and Pitres, and with Nothnagel and Exner's volumes on the localization of cerebral diseases, goes to show that we must expect more progress from further exact clinical studies supplemented by autopsies than from physiological experimentation, which of course has its place and value in the complex study of the brain, which we are just opening up.

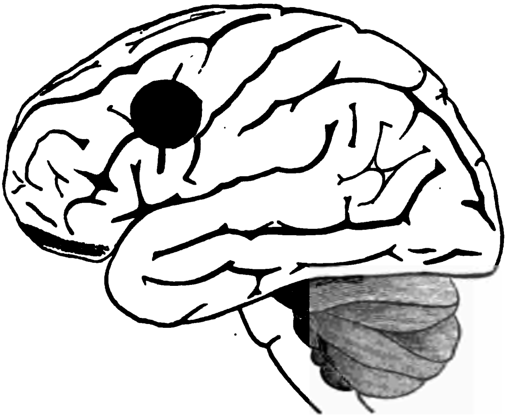
THE MOTOR CORTICAL CENTRES.

The Facial Centre.—Three instructive cases have been reported during the year. The first, by Drummond,[7] is especially interesting, as it strengthens the conclusions of human pathology *vs.* experiments upon animals. A female, æt. 29 years, admitted with pleurisy, developed empyema and repeated attacks of localized pneumonia. Operation for empyema. Three months after admission convulsions occurred, some of which were accurately observed by the house surgeon. The spasm began in the right eyelid, conjugate deviation of eyes to right, followed at once by deviation of the head to the right, twitching of the nostril, drawing up of the right angle of mouth (limbs still flaccid), rigidity and spasm of the right arm, rigidity and spasm of the right leg; then spasms in left side of face and limbs; general spasm. There were loss of consciousness and dilated pupils; repeated attacks, followed by conjugate deviation to left, and paralysis of right arm and leg. As it was probable that an abscess had formed in the left hemisphere, an operation was performed by Mr. Waldy, which resulted negatively, no abscess being seen or pus withdrawn by a hypodermic syringe. At the autopsy the source of failure was demonstrated. The operation had been made over the foot of the third frontal gyrus and the basal ends of the pre- and post-central gyri. The abscess was found in the base or caudal part of the second frontal gyrus where it becomes confluent with the pre-central. The causes of error in this operation seem to us to be, first, a strict belief in Ferrier's doctrine that the facial centre adjoins the speech centre, and, second, faulty projections upon the patient's skull. If the induction from human cases had been strictly followed, and the caudal part of the second frontal gyrus sought for by means of Broca's projections, the abscess would have been easily found, and probably, as indicated by the results of the autopsy, the patient's life saved. The figure shows that the operation was so executed as to reach the centre for speech and lingual movements. A secondary source of error was faith in Ferrier's notion of the centre for movements of the ocular muscles, possibly correct in monkeys, but as yet wholly unjustified in man. The second case, by J. J. Putnam,[8] was that of a female, æt. 38 years, whose illness of twenty-one months was characterized chiefly by intense headache, gradual abolition of speech, and paralysis of the right buccal muscles. The autopsy revealed a large sarcoma, super-



ROUNDED SPACE, SEAT OF ABSCESS. SQUARE SPACE, PLACE WHERE ABSCESS WAS SOUGHT.—(*London Lancet.*)

ficially situated, compressing the caudal ends of the second and third frontal gyri of the left side and extending under the frontal lobe. This case by itself is not of great value for localization study, but taken along with others it has weight. We know the function of the base of the left third frontal gyrus exactly; we also know that lesion of the orbital gyri produces no marked symptoms; and we may thus refer the buccal paralysis on the right side to the lesion of the caudal end of the left second frontal gyrus. The third case is by the reviewer.[9] A boy æt. 7 years, after a mild attack of measles, showed paresis of the right cheek with dribbling of saliva from the angle of the mouth, and paresis of the right arm. No aphasia. A few days later there occurred an attack of clonic spasm, limited to the right cheek, with suspension of speech and deviation of eyeballs to right. This monospasm continued for more than two hours. After this attack there remained marked paresis of cheek, and slight weakness of right hand; no aphasia or choked disk. For five or six months right hemiparesis remained, much more marked in the cheek; no return of spasm. Occasionally there was some (subjective) numbness, with formication in right hand, beginning in forefinger. It is to be noted that although numbness several times occurred first in the right hand only, the spasm and the paresis were most marked throughout in the lower facial muscles. In April of the next year, 1881, the child died of tubercular meningitis. The autopsy revealed the signs of this fatal illness, and also a patch of simple adhesive meningitis, intimate adhesion between the inner aspect of the dura-mater and the pia over a round area about 25 mm. in diameter (evidently of ancient formation), covering the caudal extremity of the left second frontal gyrus and a part of the pre-central at this level. There was no appreciable lesion of the cerebral substance.



PATCH OF LOCALIZED ADHESIVE MENINGITIS OVER THE SECOND FRONTAL AND PRECENTRAL GYRI OF THE LEFT HEMISPHERE IN CASE 1.—(*Am. Jour. Med. Sci.*)

Centre for Lateral Movements of the Eyeballs.—In favor of the existence of such a centre in the frontal lobe, only one case is reported, by Delbet,[23] *vide* summary under Brachial Centre. The lesion in this case is too large and its topography too loosely described to give it much weight as against several negative cases in which the movements of the eyeballs remain normal in spite of large destructive lesions of the gyri in front of the pre-central gyrus.

The Brachial Centre.—Salessi[10] reports the case of a male æt. 32 years who died of dementia paralytica. Nineteen years before he had suffered amputation of the right arm. Among other lesions there was found at the autopsy atrophy of the upper part of the left pre-central gyrus. Second case: Dr. Joseph Coates[16] relates an interesting traumatic case. A male adult received a compound fracture of the left fronto-temporal region which, after removal of bony fragments, left an aperture three inches long by one inch wide above and one half an inch below. Two cerebral hernias were removed. As symptoms there were almost complete and permanent paralysis with contracture of the right arm; temporary paralysis of the right face and lingual deviation to right; paresis of right leg; more or less aphasia, purely motor. No anæsthesia (muscular sense good). Later he had convulsive attacks beginning in the right cheek and arm, leg, and whole of right side, also others with loss of consciousness, in which general convulsions followed the right-sided spasm. After healing of the wound there remained only marked paralysis with contracture of the right arm and partial motor aphasia. The principal scar extended from above the middle of the left eyebrow to the coronal suture. Evidently the middle portion of the pre-central gyrus was injured, though the second frontal perhaps the third were also torn. The case is perhaps of most value as against the existence of a cortical motor centre for movements of the eyeballs in the second frontal gyrus as claimed by Ferrier and by Horsley. A third case, by P. Delbet,[23] is one of abscess secondary to chronic empyema in a man æt. 24. Sudden onset of brain symptoms by a general epileptic attack (tongue bitten), followed by several localized seizures involving the lateral ocular muscles (deviation to the left, in spite of patient's efforts to look the other way), and the flexors of the left arm. No facial spasm noted; pupils normal; speech slow.

Left arm paralyzed and somewhat contractured. Death on eleventh day with high temperature. Autopsy showed an abscess, wholly subcortical, under the posterior extremity of the first frontal gyrus as far as the fissure of Rolando. The ventricles were filled with pus. From the not-very-clear topographical description, it would appear that the lesion was clear above the facial centre, and involved chiefly the centre for the upper arm. Its anterior extremity was 2 cent. in front of the pre-central sulcus, but its relations to the second frontal gyrus are not given.

A fourth and very instructive case, almost equivalent to an experiment, is by Bottard,[26] of Havre. A healthy laborer æt. 22 years was struck on the right side of the head by a falling brick, which made a triangular compound fracture of the skull above and behind the ear. After momentary loss of consciousness it was found that the left arm was completely paralyzed and slightly anæsthetic. No other paralysis until later, when symptoms of meningitis set in. Trephining was put off until the eighth day, when, the patient being comatose, the operation was done! Death on the eighth day after operation. The autopsy showed, besides general purulent meningitis, a large contused and lacerated wound of the right hemisphere, involving transversely the upper (?) third of the pre- and post-central gyri, extending from the base of the first frontal to the first or second parietal gyri. Between the top of the brain (edge of median fissure) and the lesion there was healthy substance 2.5 cent. (1 in.) wide. The injury to the brain was therefore exactly in the centres for the shoulder and arm. It is probable that this life would have been saved by a timely thorough operation.

The Crural Centre.—Case IV. of Hun's valuable paper[11] furnishes a striking piece of evidence in favor of the existence of a cortical motor centre for the leg. A female, aet. 42 years, a year after a fall in which she struck the back of her head on the sidewalk, began to have epileptiform attacks beginning always in the left foot, affecting the whole left side, with loss of consciousness. Later there developed left hemiparesis, with tactile anæsthesia and paresis in face, arm and leg, double neuro-retinitis. At a later stage the paralyzed left limbs were flexed and somewhat rigid. Autopsy showed a hard ovoid tumor (37 by 30 mm.) upon the upper extremity of the post-central gyrus near the longitudinal fissure. This lesion is placed exactly as was one in a case reported by the reviewer,[12] in which the first convulsive movement appeared in the opposite foot (toe). König[13] reports the following case: Male, aet. 35 years, who had ordinary epileptic attacks after a fall in water. Two years later the attacks became peculiar, being preceded by an aura (numbness) in the left foot and in left arm, and paresis of the arm and leg developed with spasm of the leg only. The face was neither paralyzed nor convulsed (?). In some attacks there was loss of consciousness: this always occurred when the convulsive movements extended to the opposite (right) side. Patient also had simple vertiginous attacks. There was no choked disk. The whole left side was less sensitive to pain than the right; muscular sense intact. The autopsy showed a tumor involving the whole of the right paracentral lobule; the pre-central gyrus was hypertrophied (?) to double its normal size and compressed the post-central. The tumor was a glioma. Nothing is said of the histology of the enlarged pre-central gyrus. The case is not satisfactory in many respects, and is of value only in connection with others. The *signal-symptom*, as we would propose to call the aura or first localized spasm in cases of localized cerebral lesion, was, however, clearly present, and pointed to the leg centre. A third case is by the reviewer.[14] A male, aet. 49 years, non-syphilitic. Symptoms: paresis of right leg (signal symptom), clonic spasm in right abdominal muscles; right-sided epileptiform attacks, with spasm first in abdominal muscles, then in leg, next in arm, none in the face; paralysis of the right leg, paresis of right arm, no aphasia except two days before death, no choked disk, no headache. The leg was much more paralyzed than the arm, and in this member the loss of power was more marked in the shoulder and upper arm. No anæsthesia. The autopsy revealed a subcortical tumor in the upper (mesal) ends of the pre- and post-central gyri, or, in other words, dorsad of the paracentral lobules. The tumor was a globular sarcoma, about 25 mm. in diameter, with two minute tumors lying below it in the white substance. No other lesions in the brain. A fourth case is case VII. of Horsley's essay,[1] published in full in the *British Medical Journal*, Oct. 10, 1886. Epileptic seizures followed a blow on the top of the head, the signal symptom being flexion of the great toe. An operation revealed a thick cicatrix adherent to the gray matter at the upper end of the fissure of Rolando; its excision cured the patient. A fifth case is number X., in the same paper, in which a large diffused tumor of the upper motor area was removed, and with it a portion of the cortex in front of the upper end of the fissure of Rolando. The result was permanent complete paralysis of the opposite great toe. Sixth case, by Delas.[15] A man, aet. 43, with residue of infantile poliomyelitis in left lower extremity. Seven months before admission had repeated convulsions in the right lower extremity, beginning by flexion of the great toe, and followed by paresis and anæsthesia (?) of the member. On admission presented the usual atrophy and deformity of poliomyelitis in the left lower extremity; in the right there was some loss of power, but no atrophy; could not stand. Some incoördination of right leg and arm. A month later localized convulsions affected the right upper extremity, starting in the shoulder muscles. In one attack loss of consciousness. Later right hand paralyzed and anæsthetic. The autopsy revealed a tumor as large as an orange, involving the left paracentral lobule and the upper part of the pre- and post-central gyri. The white substance beneath the tumor was softened and yellowish. Tumor was an encephaloid sarcoma. Seventh case, by Hughlings Jackson.[17] A woman, aet. 52, suffered from suddenly developed paralysis of the left leg, without anæsthesia, but with increased reflexes. At the autopsy the left femur (fractured before death) was found cancerous, and there were also found cancerous growths in the brain, one in the posterior extremity of the marginal convolution (first frontal, adjacent to paracentral lobule).

Questionable Motor Centres.—No contribution has been made bearing on the location of a *laryngeal centre* which, judging from three or four older observations, may be situated (in right-handed persons) in the caudal extremity of the right third frontal gyrus, homologous to the speech centre in the left hemisphere.

Centre for lateral cervical movements (*torticollis*).—One case without autopsy is recorded by G. V. Poore.[22] A torticollis to the right (not definitely described) existed for six months in a male adult. There was also a scar on the left side of the head over the posterior part of the superior and middle frontal gyri, and the patient had had syphilis. After section of the left sterno-mastoid muscle, it was discovered that the right splenius was mostly active. A course of mercury cured the patient. Dr. Poore and Dr. Broadbent considered the case as analogous to cases of torticollis in conjugate deviation.

Centre for Masticatory Movements.—No decided progress has been made in this direction. V. Langer[144] summarizes a number of observations, by various authors and himself, of trismus and of paralysis of the muscles of mastication, in which widely different lesions were found. In two of the cases, however,—one by the author, the other by R. Lépine,—there were hemiplegia and trismus during life, and in both the autopsy revealed lesions of both insulas; in one case hæmorrhage, in the other softening from thrombosis of the sylvian artery. In studying this question it is well to bear in mind that, according to Broadbent's law (considered as applicable to the muscles of mastication by Exner), the muscles in question receive a strong bilateral or automatic innervation, and are probably not largely represented in the cortex.

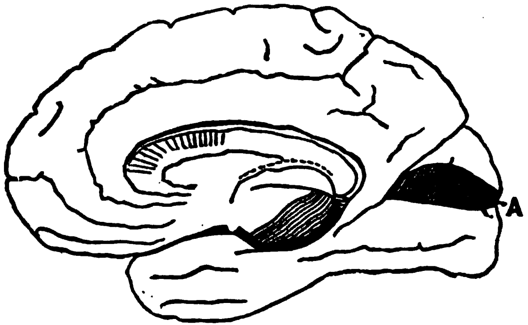
Centre for Lingual Movements.—A greatly to be regretted confusion has taken place in the study of this centre between cases in which the intrinsic and those in which the extrinsic lingual muscles are paralyzed. The former cases include cerebral pseudobulbar paralysis, in which coarse lingual movements are impaired or abolished, and cases of motor aphasia, in which the delicate movements of speech are lost. The lesion causing the aphasic state is well known to be in the caudal extremity of the third frontal gyrus, on the left side in right-handed persons.

Centre for Coarse Lingual Movements.—An example of paralysis of the extrinsic lingual muscles on one side is reported by Bernheim.[24] A girl, æt. 23 years, presented multiple sarcomata. In January, 1886, well-marked deviation of the tongue to the right occurred, and persisted until death, a month later. There was no facial or other paralysis, and no aphasia. The autopsy showed numerous sarcomatous tumors throughout the body; in the brain there was a small blood-cyst with sarcomatous walls, 5–6 mm. in diameter, located toward the inferior borders of the lower extremity of the left pre-central gyrus, on its anterior surface, about 6 mm. behind the sulcus which separates it from the third frontal. The muscles paralyzed by this lesion would be (probably) the stylo-hyoid and the stylo-glossus, which, together with the genio-hyo-glossus, are the most active factors in protrusion of the tongue. Examples of paralysis of the extrinsic and intrinsic lingual muscles by a cerebral lesion are recorded by Drummond.[25] (1) Besides glossoplegia, there was right hemiplegia, face included. The lesion consisted in thrombosis of the left lenticulo-striate artery, and consequent softening of the posterior part of the caudate nucleus, the anterior extremity of the thalamus, and the intervening portion of the internal capsule. The value of the case is greatly reduced by the absence of any statement as to the gross and microscopic state of the medulla oblongata. As the patient was fifty years of age, it is very possible that small arteries in the bulb were diseased and thrombosed.

Negative Localization Cases.—L. Brieger[18] reports a case of left hemiplegia caused by a large cerebral tumor (sarcoma pia matris) involving the first and second frontal gyri, the pre- or post-central gyri, and extending to the parietal lobe in the right hemisphere; also exerting pressure upon the inner aspect of the left hemisphere in the region of the first frontal. There were typical hemiplegia with contracture of the arm and leg and only slight tactile anæsthesia of the left arm; no aphasia, or choked disk. It is expressly stated that the movements of the eyeballs and the pupils were normal. This is against the existence of a centre for ocular movement in the frontal lobe. A second case against the existence of such a centre has already been referred to under the heading of "The Brachial Centre," case second, *vide* p. 9. Féré[19] reports to the Société de Biologie a case of tumor of the apex of the left frontal lobe which had caused only blindness and convulsions. A fourth case by Alexander[20] had presented no brain-symptoms during life; at the autopsy a tumor was found involving and destroying all the orbital convolutions and the olfactory nerve on the right side; it pressed upon the opposite hemisphere, and depressed the roof of the orbit. A fifth case by de Schweinitz[21] is more complicated. A strumous child (articular and glandular lesions), aged 8 years, suddenly developed general convulsions, more marked on the left side, followed by right hemiplegia. Convulsions recurred for thirty hours, when death occurred. The convulsions began on the left side, with twitching of the eyelid, and face, neck, arm, and leg, passing over to the right limbs. The eyes and head were turned toward the left side. There was double optic neuritis. The autopsy revealed an enormous (3½×2⅓ in.) tumor involving the right frontal lobe from the base of the third gyrus to the orbital gyri, extending back so as to involve two or three folds of the island of Reil. The tumor penetrated into the white substance and destroyed the head of the nucleus candatus. The right hemiplegia was fully explained by finding the left crus cerebri in a state of recent hæmorrhagic infiltration. Hun[11] reports a case of tumor of the lower (orbital?) part of the right frontal lobe, near its middle, as large as a small hen's egg, and surrounded by a narrow zone of yellow softening. No motor, sensory, or aphasic symptoms were present; only anæmia, debility, and coma. Journiac[69] presented to the Anatomical Society of Paris an angeiolethic sarcoma of the right frontal lobe which had produced no special symptoms,—dementia chief symptom. The tumor was 6 cent. (2¼ inches) in diameter, in the outer basal part of the frontal lobe.

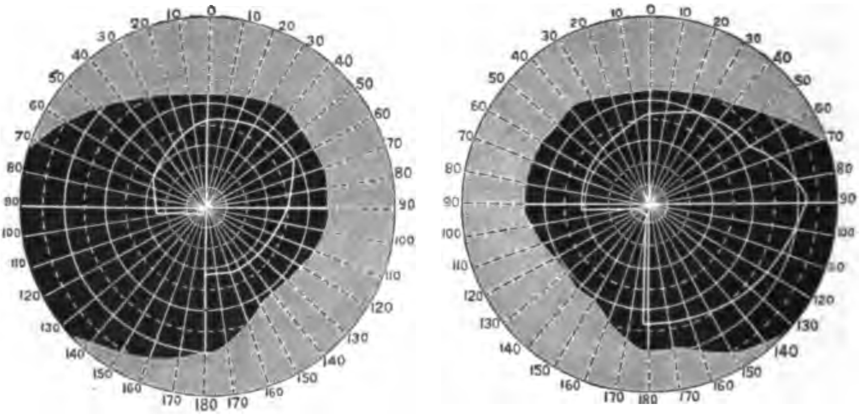
THE SENSORY CORTICAL CENTRES.

The Centre for Vision.—Henry Hun[11] has published in a paper which is otherwise very interesting, a most conclusive case proving that the cuneus in either hemisphere is the half-centre for perfect vision. During life there was observed a defect in the fields of vision involving the lower left quadrant of each field. In other words, one-quarter of each field of vision was dark, in a lateral or homonymous way. Between December, 1884, and March, 1885, the fields were mapped out several times and found unchanged. There was no distinct paralysis or anæsthesia, but there were various mental symptoms,—confusion, inability to recognize objects and to locate himself; memory was feeble; angina pectoris developed. Besides a cardiac lesion, explaining the angina, there was found a patch of softening in the right hemisphere, "strictly limited to the lower half of the cuneus." The white matter underneath was



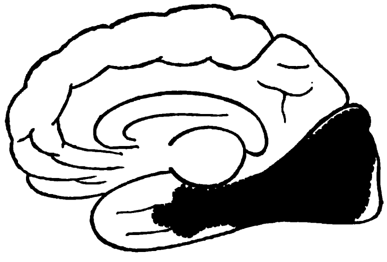
Median Aspect of Right Hemisphere. (Ecker.) A, Point of Attack.—(*Am. Jour. Med. Sciences.*)

softened to a depth of about half an inch. There were no other lesions or signs of secondary degeneration in the brain. The optic tract and nerves appeared normal to the naked eye. This unique case, in our opinion, confirms in the most positive manner the law previously propounded that in each cuneus is a cortical centre for half-vision, as proved by five recorded cases of limited superficial lesions. Anatomically, the case teaches that the fibres from the right upper quadrants of each retina are continuous with the lower half of the right cuneus. It follows that the right lower quadrants of each retina have a connection by optic fibres with the upper part of the right cuneus, and perhaps with the first occipital gyrus (Nothnagel).

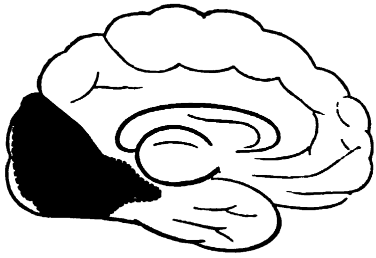


FIELDS OF HUN'S CASE: QUADRANT OBSCURATION.—(*Am. Jour. Med. Sciences.*)

A second case, in which hemianopsia was the chief symptom of a long illness, is reported by Birdsall and Weir.[27] The patient, an adult male, in August, 1885, had vomiting and slight staggering gait. There was only one severe attack of vomiting. Diplopia for distant objects next occurred and continued with intermissions. Frontal headache and a disagreeable feeling akin to numbness in the right extremities were present. He consulted Dr. Seguin, October 7, 1885, who discovered left lateral hemianopsia, not involving the points of fixation, and who made a diagnosis of tumor of the mesal aspect of the right occipital lobe, involving primarily the cuneus, and extending thence downward upon the tentorium. In November choked disks appeared under the reviewer's observations, and both externi were paretic. The case passed under the care of the late Dr. T. A. McBride, and finally into that of Dr. Birdsall, in October, 1886. There were then typical left lateral hemianopsia, choked disks; no distinct paralysis, though there was an uncertain awkward gait, not ataxic, and not typically cerebellar. Headache was rare and slight, mental action good; weakness and difficulty in standing and walking increasing rapidly, the patient consented to an operation, which was recommended by Dr. Birdsall and, in consultation, by Drs. Janeway, Spitzka, and Seguin. On March 9th, Dr. Weir, after a most difficult and admirably executed operation, removed an enormous tumor, measuring 2¾ × 2½ inches, and weighing 5½ ounces. It lay, as was expected, upon the mesal aspect of the right occipital lobe, resting against the falx cerebri, and pressing upon the tentorium. It had forced the cerebral tissue forward and downward so as to make room for itself. It was encapsulated, and the brain-tissue appeared extremely anæmic, but uninjured. What seemed to be a pedicle of the tumor lay deep down in the wound, near the junction of the falx and tentorium. Secondary hæmorrhage occurred from this point during the night, and in spite of attempts to find the bleeding vessel, etc., death took place at 2 A.M. This case, besides being of value for topographical diagnosis, teaches that an operation for brain-tumor should be made early,—as early as the medical diagnosis is established. Third case by Glynn.[28] An old woman suddenly attacked with paralysis of the right side, accompanied by complete hemianæsthesia. The eyes and head were somewhat turned to the left. Although the patient was so weak that all the special senses could not be examined, the author satisfied himself that there was right lateral hemianopsia. There was no aphasia. After death there was found a hæmorrhagic focus, measuring ¾ × 1 × ½ inch, destroying the outer two-thirds of the left thalamus and the whole width of the internal capsule behind the genu. The posterior two-thirds of the caudal division of the capsule were destroyed. This would include the optic fasciculus, yet the author naively remarks that "it is impossible to define the relation of the lesion to the sight troubles." A fourth case by L. Bouveret,[29] of Lyon, is remarkable as being one of apoplectic double hemianopsia, or complete blindness. This was the chief symptom in the case, accompanied by loss of memory and much mental confusion. The patient assisted himself with his hands, showing very perfect motility and muscular sense. Unfortunately the senses of smell and taste were not examined. The autopsy showed, as had been diagnosticated during life, patches of softening on the inner aspect of each occipital lobe, destroying the cuneus, but also invading to a certain extent the first and second occipito-temporal gyri. There were no other lesions in the brain. The softening was due to plugging, probably by embolism, of the occipital artery. Besides being an additional proof as to the location of the chief visual centre, this case is of importance because it effectually disposes of the theory which would explain the preservation of central vision in ordinary cases of lateral hemianopsia by supposing that fibres from the macula terminate somewhere in the outer surface of the occipital cortex. A fifth case was reported by Leyden,[30] in a discussion at the Verein für Innere Medicin of Berlin. Hemianopsia was observed in a patient suffering from hemiplegia. Death after a second apoplectic attack. Lesion was a tumor involving the basal and mesal aspects of the left occipital lobe; convexity normal. The tumor penetrated into the white substance. In a paper read before the Medical Society of London, November 21st, Dr. Ferrier,[31] reports three cases of cerebral disease, in which hemianopsia (lateral, we presume) coexisted with symptoms of tumor and other lesions. The details accessible to us are too meagre to allow of a useful abstract of the cases.



BOUVERET'S CASE.—MESAL ASPECT OF RIGHT HEMISPHERE : LESION SHADED. —(*Lyon Medical.*)

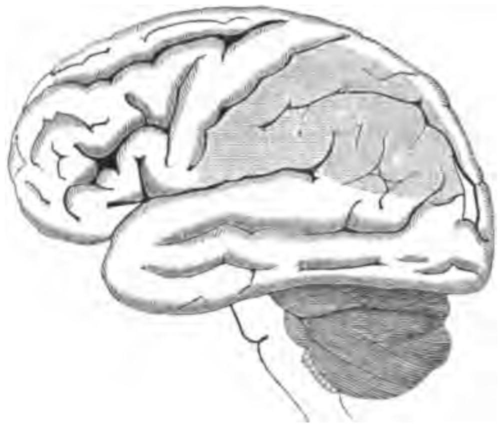


BOUVERET'S CASE.—MESAL ASPECT OF LEFT HEMISPHERE : LESION SHADED. —(*Lyon Medical.*)

The Auditory Words Centre, vide *Aphasia.*

The Centre for Muscular Sense.—No contribution to the study of this centre has been published during the year. From a study of former cases it seems probable that this mode of sensibility is centered in the inferior parietal lobule (this view was first formulated by the reviewer[32] and endorsed by Nothnagel more recently). In one of Hun's cases, however, the lesion included this centre. In this case, along with right hemiplegia, alexia, and agraphia, there was noted a remarkable awkwardness of the partially paralyzed right arm. The autopsy showed softening involving the post-central gyrus, and also the whole of the inferior parietal lobule comprising the supramarginal gyrus, and almost the whole of the angular gyrus. Most unfortunately the muscular sense was not separately or exactly studied.

Centre for Olfactory Sense.—Frigerio[33] is quoted as relating a case of "systematized delirium" with a constant perversion of the sense of smell. Among other lesions there was found atrophy of the left pes hippocampi. The observation is not worth much, but perhaps deserves record. T. W. Griffith[34] relates the following case: Male patient, 35 years of age, showed emaciation, asthenia, and polyuria. There was very slight paresis of the left side of the face. The principal symptoms were left lateral hemianopsia and progressively developed anosmia of the right nostril. No paralysis of the ocular muscles or iris is noted, and it is stated that the ophthalmoscopic examination was negative (?). A tumor was found at the base of the brain, consisting of a mass of tissue lying just external to the right optic tract and the posterior part of the right optic nerve. From a point 3 lines (6 mm.) back of the commissure the right optic tract was normal; beyond that, it was soft and of a fleshy color. The surface of the crus was involved, and there was marked pressure upon the right uncinate convolution, which was eroded to the depth of one-eighth of an inch (3 mm.). The tip of the uncus was unaffected.



OUTER ASPECT OF LEFT HEMISPHERE. SOFTENED AREA SHADED.—(*Am. Jour. Med. Sciences.*)

VARIOUS LOCALIZED ENCEPHALIC LESIONS.

(1) *Lesions of Central Ganglia: Thalamus, Nucleus Caudatus, and Nucleus Lenticularis.*—Cases of this category are negative as regards direct symptoms and the possibility of diagnosis during life. Boisvert[63] relates a case in which facial paralysis was explained post-mortem by a patch of softening at the foot of pre- and post-central gyri. There were also patches of red softening in various other gyri. The right thalamus was enlarged and contained an old hæmorrhagic infarct of ochrous color. The adjacent internal capsule was normal. There had been no symptoms which could be referred to this lesion; vision and general sensibility normal.

Jonathan Hutchinson[64] reports a case of bilateral disease of the corpora striata. Symptoms: relaxation of sphincter ani and incontinence of urine not dependent upon local conditions; utter mental prostration and physical inertia; change in temper, from austerity and precision to effusiveness and jocularity. No paralysis or anæsthesia could be detected; the knee-jerk was good; eyes normal. Rapid emaciation, stupor and coma ended the scene. Lesion: destruction by sarcomatous disease of the anterior and inner parts of both corpora striata (nuclei candati) with some hæmorrhage on the right side. The general "weakness" may have been symmetrical paresis from the symmetrical lesion.

(2) *Lesions of the Crus Cerebri.*—Alexander,[55] of Aachen, reports the following interesting case of crus lesion. Male, aged 45 years, when first seen in 1885; had contracted syphilis in 1865, followed by pharyngitis; had thorough treatment and no symptoms occurred afterward in self or family. In 1883, suffered from headache and vertigo. Soon afterward had an apoplectic attack at table, followed by paresis and numbness of the left limbs and drooping of the right eyelid, paralysis of the recti supplied by third nerve, but not of the iris, which acted normally in both ways. Was given a thorough treatment by mercurial inunctions and baths at Aachen without improvement. After spending the winter of 1885–6 in Italy, the patient returned to Aachen much worse, with mental depression, more marked paralysis and anæsthesia on the left side of the body, and total paralysis of the right motor oculi (ophthalmoplegia interna et externa). The autopsy showed

thickening of the basilar artery and many of its branches; an area of softening in the deeper part of the right crus cerebri, extending upward and forward to the thalamus and floor of the third ventricle. During the illness there were no impairment of vision, hemianopsia, or choked disks. The author indorses Mauthner's wholly theoretical assumption that the ciliary fibres of the third nerve arise from a separate nucleus in the floor of the third ventricle, far forward of the main nucleus for the nerve. There is, it seems to us, another and more practical lesson in this case. This patient was treated *à la mode* Aachen, only with inunctions of mercury and baths; there is no mention of the use of iodide of potassium, which we believe, if used according to the American method would have arrested the disease in its first stage and prolonged the patient's life indefinitely.

(3) *Lesions of the Pons Varolii.*—Bruno[40] relates the following case of tumor:—Child æt. 2½ years, having an intense double otitis media pur. of tubercular nature, suddenly developed paralysis of the left sixth nerve, with weakness of the right internal rectus, anæsthesia of the cornea, and conjunctiva most marked in the left side (no analgesia of rest of trigeminal distribution); paresis with contracture and increased reflexes in the right extremities, without anæsthesia. Later somnolence, general weakness, choked disks. The lesions were: A few small isolated tubercles in the cerebral cortex; a large tubercle occupied almost the whole of the tuber annulare, bulging into the fourth ventricle, 3 cent. in front of the calamus scriptorius. Some normal nervous tissue lay to the right of the tumor in the pons. This tumor evidently at first lay in the left half of the bulbo-pontic region, destroying the nucleus of the left sixth nerve, and producing crossed paralysis. The question of abscess from the aural disease might have been brought up in this case and would have been insoluble until after death.

Hun[50] has made an excellent study of a case of gliometous hypertrophy of the pons. A child, æt. 6 years, of a family prone to nervous diseases, presented the following symptoms:—pain in the top of the head on coughing, an awkward, unsteady gait, occasional vomiting, speech drawling, mouth open much of the time, drooling, and some dysphagia. Head drawn toward right shoulder, stands with feet wide apart, the right leg is more rigid than the left, and she takes smaller steps with it; hence a tendency to walk circularly to right. No anæsthesia of face or body, knee-jerk exaggerated on both sides; double optic neuritis. Later, the diffused paresis increased, speech became indistinct. Toward the close of life the child's head enlarged, although there was no headache except a few days before death (occipital). At no time convulsions. Mind clear until death which occurred about five months from onset. The symptoms throughout were not those of irritation, but of gradually failing functions of bulbar and pontic nervous elements. The autopsy showed, besides flattening of cerebral gyri and distended ventricles, an hypertrophied (three or four times its normal size) pons variolii, which microscopic examination showed to be a gliomatous infiltration, pretty nearly limited to the pons.

Griffiths,[51] of Cardiff, case of tumor with crossed paralysis. Child, æt. 4½ years, admitted to hospital January, 1885. Nine months before, fall on the head, followed by drowsiness and vomiting; vomiting continued every morning for three months. Five months later paresis of right external rectus and weakness of limbs. On admission child presented, paresis of whole of right side of face, paralysis of right abducens, slight paresis of left extremities; approach to De R. in right facial muscles; double optic neuritis. Lesion: a reddish tumor, size of hazel-nut projected from the basal surface of the pons on the right side; surrounding nervous tissue soft. Microscopic examination showed tumor to be tubercular. Nodules of caseous pneumonia in both lungs, and some bronchial glands were caseous. The same author reports another case of crossed hemiplegia in an adult male, due to softening produced by thrombosis of the basilar artery. After headache lasting a few days, the patient had a nocturnal "fit," followed by paralysis of the right side and loss of speech. When seen there was left facial paralysis, right-sided body paralysis, with some anæsthesia; the pupils small with doubtful light reaction. No paralysis of ocular muscles; upper facial muscles normal. Later, dysphagia, hiccough and difficult breathing. The right-sided anæsthesia involved the face. A prominent symptom was abnormal emotion (fits of sobbing). The left half of the pons was the seat of greatest softening. As the patient was unable to answer questions it remains uncertain whether he had had syphilis; yet, he was rather young (33 years) for ordinary "atheroma," which was found in the basilar and other arteries at the base of the brain. Marfan[52] reports the following case of hæmorrhage. Woman, æt. 58 years, previously in good health, suddenly fell in the street without convulsion or loss of consciousness. When examined in the hospital she presented paralysis of the right abducens nerve, total paralysis of the right face; a certain amount of glossoplegia with loss of speech (understanding everything); paresis of left arm and leg with hemianesthesia on the same side, face included; special senses not involved. Death occurred on the tenth day. The autopsy justified the diagnosis of hæmorrhage in the pons; the right half of this organ containing a clot, which passed the median line by about $\frac{1}{5}$ inch. Some valuable topographical statements are given in the original. [It is important to note that in the two cases above analyzed, one of softening, the other of hæmorrhage, there was no conjugate deviation.] Another case of bulbo-pontic hæmorrhage is recorded by Dutil.[53] Male, æt. 54 years; apoplectic attack, followed by paralysis of right extremities and of the left face (upper muscles included). There was slight anæsthesia on the right side. The body hemiplegia improved, but both sixth nerves (abducens) were paralyzed. Death on thirteenth day. Lesion: clot size of hazel-nut in the dorsal levels of the medulla-pons transition, with destruction (softening?) of the floor of the fourth ventricle at one small spot. The clot was in the location of the left eminentia teres, and involved both nuclei of the left facial nerve, and the nuclei of both sixth nerves. The two sixth nerves, and the left facial showed microscopic signs of degeneration. It is noted that during attempted turning of eyes to one side the associated rectus internus did not contract, although the patient could converge well with both eyes; hence the case supports the hypothesis of crossed associated action of the sixth and third (filament to internal rectus) nerves.

Bourneville and Isch-Wall[59] report at great length a case of tubercular disease of the pons in a child aged 5 years. The symptoms were very complex and obscure and do not allow of condensation; but the case will repay perusal and the authors have added a summary of ten other cases of this rare lesion by different authors.

(4) *Lesions of the Medulla Oblongata.*—Case of tumor in the

medulla, by Dr. Glynn.[48] Male, æt. 31 years, admitted to hospital March 18, 1887, complaining of loss of power in arms and legs and difficulty of breathing. Never syphilis. The tongue felt numb and swollen; vomiting had occurred; there was no headache. Examination showed a diffused loss of power with increased cutaneous and abolished patellar reflex; normal vision, with normal pupils and slight optic neuritis; smell, taste and hearing normal; slight tactile anæsthesia of limbs; speech thick, and swallowing a little difficult. Speech grew worse, expectoration inefficient, there was paresis of the lips and cheeks, pain in the legs, increasing weakness of muscles of all extremities and trunk, to almost complete paralysis. Death occurred on March 24 after a slight convulsion, preceded by extreme bulbar symptoms, including a trace of sugar in urine, and rapid pulse. The respiration for a time was only costal. The only facial anæsthesia was on right side, where pricking was poorly felt. Autopsy revealed a gliomatous tumor not larger than a pea, placed in the middle line of the medulla at the level of the calamus scriptorius, and $\frac{1}{8}$ inch below the surface of the fourth ventricle. A diagnosis of bulbar lesion was made during life.

Oppenheim[56] reports the following extraordinary case of chronic bulbar paralysis without anatomical lesions. Female, aet. 29, observed in 1885–1886. Symptoms: weakness of arms and legs, difficult speech and deglutition; lips nearly powerless; paresis of palate, with nasal speech and regurgitation of fluid through the nose; respiration labored and voice weak; no muscular atrophy, abnormal reactions, or anæsthesia. Death after intense dypsnœa two years after onset of disease. Lesions:—Pneumonia from food infarctions; medulla oblongata and nerves normal to naked eye, and microscopical examination confirmed this unlooked-for result. The author refers to two similar cases, one by Westphal, the other by Wilkes. In reply to an inquiry he stated that the brain appeared normal to the naked eye, and that the arterial disease which usually causes cerebral pseudo-bulbar paralysis was not present. [Two capital symptoms of chronic bulbar degeneration are not mentioned and probably were wanting, viz., inability to protrude the tongue and shrivelling of that organ and salivation.] In an elaborate paper Oppenheim and Siemerling[57] treat of pseudo-bulbar paralysis, and acute bulbar paralysis, illustrated by five cases. General semeiology: apoplectic attack with or without loss of consciousness, paralysis of the extremities, with bulbar disorders of speech, deglutition and respiration; often also, optic nerve atrophy, apathy or dementia; course of disease remittent. Lesions: arterio-sclerosis of numerous arteries; areas of softening in the internal capsule, the basal ganglia, the corona radiata (once in cortex of frontal and temporal lobes); lesions often bilateral. In these cases although to the naked eye the pons and medulla appeared normal, the microscope revealed small foci in the pons, in the pyramidal tracts and under the floor of the fourth ventricle. The authors conclude that true cerebral pseudo-bulbar paralysis (cases of Boyer, Eisenlohr, Ross and others) is rare, while the mixed form in which lesions are found in various parts from the cerebrum to the medulla are far more common. The presence of muscular atrophy and the absence of eye-symptoms, aphasia and mental symptoms, strongly point to true bulbar paralysis. Oppenheim[58] calls attention to a little-known cause of atrophy of the olivary bodies through arterio-sclerosis, and dilatation of the vertebral arteries,—in some cases an aneurismal enlargement. The left vertebral artery and the left olive are more frequently the seat of lesions. In some of the cases distinct bulbar symptoms had been observed. A dilated vertebral artery may cause pressure upon the occipital nerve causing occipital neuralgia (Senator). The inferior posterior cerebellar artery may injuriously affect the vagus nerve, causing respiratory symptoms. W. Pasteur[60] relates a case of probable acute bulbar disease. A child a little over two years old, had a febrile attack, followed by complete paralysis of the right side of the face, impaired articulation, and inability to swallow. In a few days the last symptom disappeared, and later speech returned. When examined three months after attack, the only symptoms present were complete (Bell's) palsy of the right face, deviation of tongue to right, with slight wasting of the right half of the tongue. No symptoms in rest of body; child healthy. Three months later upper facial muscles were better. The author considers it to have been a bulbar poliomyelitis expending its chief force upon the right facial nucleus. The incomplete recovery is rather in favor of this view as against a neuritis.

(5) *Lesions of the Cerebellum.*—Seguin[36] reports four cases of tumor of the cerebellum completed by autopsy. Symptoms: lesion

of the optic nerve, either choked disk or secondary atrophy was present in all cases, probably as an early symptom. Headache was present in all cases; distinctly occipital and paroxysmal in three cases; occipito-frontal and never severe in Case I. (cyst of vermis superior). Vomiting was a very early symptom in three cases, usually matutinal, and not due to indigestion. The gait was affected in all cases. In Cases I. and IV. there was typical cerebellar titubation; in Case II. staggering with tendency to left; in Case III. a diffused staggering like that of intoxication (not observed by author). Motor eye-symptoms; in Case I. there occurred various types of nystagmus; in Case II. there was conjugate deviation (without vision) to the left, away from the lesion. Paralysis: slight but distinct right-sided paresis (face and tongue excepted) in Case II. The other cases presented only diffused loss of power. Ataxia, strictly speaking, was present only in Case II., affecting the right upper extremity, on the same side as the tumor. Anæsthesia was found only in Case IV., demonstrable only on the left side of the face and on left fingers, opposite the tumor. Vertigo: subjective and static vertigo was not present in any case. Psychic symptoms were absent in all cases. The lesions found were as follow: Case I. cyst about 2 inches in diameter, destroying the superior vermis except its anterior third, penetrating into the right lobe, but not destroying the nucleus dentatus, exerting some pressure upon the floor of the fourth ventricle. (There were other recent lesions in the cerebrum and pons, not at all related to the symptoms observed during the eighteen years of illness, and attributed correctly in 1878, seven years before death, to a lesion of the superior vermis.) Case II. tumor (sarcoma) occupying the inferior part of the right lobe, compressing the underlying portions of the pons. The upper three-fourths of the same hemisphere was occupied by a cyst, invading the upper and middle portions of the vermis superior. Case III. a fibro-sarcoma in the left lobe bulging out toward the anterior and inferior part of the middle lobe, and pressing upon the aqueduct of Sylvius. Case IV. sarcoma occupying the greater part of the right lobe, and exerting much pressure on adjacent parts. In Case II. the lesion was apparently quiescent, and the patient, aged 14, died of tubercular meningitis. Case I must be considered as a typical case verifying Nothnagel's law of the dependence of cerebellar titulation upon destruction of the vermis superior. In three of the cases repeated and remarkable remissions were produced by large doses of iodide of potassium.

Drummond[37] contributes an interesting case with rotatory movements. A boy, aged 7 years, had headache, vomiting, staggering gait, atrophy of optic nerves, loss of knee-jerk, glycosuria. The day before death, had two convulsions with loss of consciousness, and each time he rolled from left to right. The lesion was a tubercular tumor, as large as a walnut, in the left lobe, extending over the median line into the right; lateral ventricles distended. Marten.[42] Patient aged 52 years; symptoms, severe occipital headaches, vomiting, double optic neuritis, and tendency to fall directly backward. Autopsy revealed a cyst in the cerebellum, pressing upon the fourth ventricle. Humphrey.[43] Female, aged 21 years. For one year frontal and parietal headache, dizziness, and vomiting. Later blindness. Lesion: a round cell sarcoma in the upper part of the cerebellum; much gelatinous basal infiltration of meninges, particularly about optic commissure.

Bennet May,[44] of Birmingham, relates a most instructive case of tumor removed by operation. The credit of the correct medical diagnosis belongs to Dr. Suckling. Boy, aet. 7 years, admitted to hospital July, 1886. Had had rheumatic fever a year before for 3 months, severe frontal headache and vomiting, failure of sight. When examined he presented paralysis of the right sixth nerve with conjugate deviation to left; intense double neuro-retinitis, with almost complete blindness. The gait was staggering, chiefly directly backward, but also to the left; speech and mental action normal. A month later was unable to stand, had retraction of the head, nystagmus appeared, and the knee-jerk was lost on the right side. Dr. Suckling located the tumor in the right side of the cerebellum. Operation: after removal of periosteum, a trephine was applied on the right side of the skull, "about the centre of a quadrilateral space bounded above by the right superior curved line, below by the foramen magnum, and inwardly by the occipital crest." The aperture was enlarged by bone-forceps. The dura bulged exceedingly, and after a flap of it had been turned up the cortex of the cerebellum appeared normal, but palpation detected a hardness at one spot. After incision the hard mass could be felt at about one inch below the surface. It was easily dug out by the handle of a small teaspoon. The child died a few hours later of exhaustion. Owing to Mr. May's absence from town the operation had been deferred until extreme prostration had occurred. The author states he found the surgical procedure "extremely easy of accomplishment."[1] Laennée,[45] of Nantes, relates a case which, in several respects is valuable. Male, aet. 42 years. No syphilis; in 1878 fell from a carriage bruising chest and right side of head; a year later staggering gait developed. Admitted to hospital June, 1886. For two years previous unable to stand; walked about on all fours; there is left facial paralysis; palatal paralysis and difficult deglutition; the external rectus of left eye is paretic; pupillary action and vision good (no ophthalmoscopic examination); there is aphonia. Paralysis and anæsthesia (muscular sense preserved) are absent, but there is general inco-ordination of the entire body, and true ataxia of arms and legs. Later, slight loss of sensibility to temperature appeared in the lower limbs. The bulbar symptoms, palatal and pharyngeal paresis, aphonia, progressed rapidly; sudden death in Feb'y, 1887. Autopsy showed a tumor as large as a goose-egg, of a cheesy aspect, involving the vermis inferior and slightly invading the right lobe. Pressure was strongly exerted on the floor of the fourth ventricle. The most interesting feature of this case is the true projectile ataxia which was present. This is not a symptom of cerebellar disease, strictly speaking, but is in all probability due to injury to the dorsal aspect of the mesocephale by downward pressure. This symptom was also present in Case II. of Seguin's[36] essay, coinciding with absence of patellar reflex. In the case above analyzed the reflex was normal. Schultze,[54] of Heidelberg, contributes a rare case of cerebellar and bulbar atrophy. The patient, aet. 39, had had diabetes insipidus from childhood. No syphilis, but alcoholic excesses admitted. In 1882 appeared drawling speech, staggering gait, frequent vertigo, occasional headache and vomiting. A year later slight nystagmus, a trace of volitional tremor, increased patellar reflex; paræsthesiæ in legs, cincture feeling, paresis of bladder; no ocular symptoms, or anæsthesia. In 1886 increase of bulbar symptoms with general paralysis and death by apnœa. Autopsy showed arteritis obliterans of basal arteries, more especially of the left vertebral. The medulla and cerebellum were atrophied. The latter measured 9 cent. in breadth (instead of 11.5 to 12.5), 3.75 cent. in length (instead of 5.5 to 7.), and 5 cent. in height (instead of 6). Microscopic examination showed atrophy of medullary sheaths of nerves in the white substance, and of the Purkinje cells. In the atrophied corpora dentata were calcareous granules. The vessels were extensively degenerated. In the medulla was found primary atrophy of the pyramidal tracts and the nuclei of the vagus and hypoglossal nerves; the olives were small. Degenerative changes could also be traced in the three cerebellar processes. The ascending cerebellar tracts (of the cord) were normal. The author attributes the atrophy to the arteritis, and this to the alcoholism. Leslie and Bramwell,[41] case of a male aet. 24, seen in April, 1885. Since end of 1880 had suffered from causeless vomiting which resisted all treatment. In autumn of 1882 staggering or "lurching" gait noticed; was somnolent. Headache appeared during 1884, and became constant and severe (frontal mostly); increased staggering; failure of memory. Optic neuritis with normal pupils; skin and deep reflexes normal; no distinct paralysis. Later, convulsions occurred in night, and also local spasms in the right arm and leg; severe headache; right tinnitus (steam-sound). Death after an attack of vomiting in Nov., 1886. Autopsy revealed usual signs of cerebral compression. Cyst size of orange involved the upper part of cerebellum; the cyst involved by invasion and compression the right lobe. The middle lobe was almost entirely destroyed by a gliomatous tumor containing a calcareous nodule. The cyst had sprung from the tumor. The authors add that relief was obtained by the use of iodides, but the doses used were much too small to be of any real benefit. (Compare ref. 36 for effects of large doses.)

(6) *Lesions of the Pineal Body.*—R. Schulz[47] contributes a case of tumor of the pineal gland, and adds a tabular analysis of five other cases. Male, aged 28 years; first seen by Schulz in March, 1884. Headache began in 1879 and became severe in 1883; worst in the morning. Pain mostly occipital, with a sensation of splitting or opening of the skull; diminished vision, especially on left side. (A few weeks before first examination, obscuration of outlines of optic nerves, without actual swelling; papillæ red.) The eyes are somewhat prominent; percussion of skull reveals tenderness in occipital region; no trace of paralysis, ataxia, or loss of sensibility; the walk is somewhat staggering, and the patellar reflex is exaggerated; no vomiting, abnormality of the pulse or urine. After some improvement, dysphagia appeared, and all

symptoms were aggravated in Sept., 1884. Had some occipital pain, and splitting sensation in head; choked easily when swallowing (no lesion of larynx). In Dec., 1885, patient lay in bed with head extremely flexed on chest, congested face, injected eyes; eyeballs prominent; pupils normal; no paralysis in head or body; no anæsthesia; slight paræsthesia in feet and right arm; bladder normal; can take liquid food only through a tube; on both sides increased patellar reflex and ankle-clonus. Excessive headache and sleeplessness. Death on Dec. 25, after injection of 0.01 ($\frac{1}{6}$ grain) of muriate of morphia. Autopsy: ordinary signs of pressure in cerebral convolutions and white substance; ventricles greatly distended; tuber cinereum and optic chiasm bulge and fluctuate. The pineal body is transformed into a grayish and vascular tumor as large as a walnut, lying upon the corpora quadrigemina. The diagnosis during life was tumor of the brain, location unknown. Pontoppidan.[61] Male, aged 31 years, who contracted syphilis two years before appearance of illness. Symptoms: disposition to lie on left side; peculiar gait; *i.e.*, when told to walk forward would step two or three times on the same spot, then walk backward. There was inclination to turn to the left, and also to stoop to the left; later, tendency to rotate on longitudinal axis to the left. When standing was inclined to fall backward. Neuro-retinitis was present. At the last, sopor, profuse perspiration, opisthotonos, conjugate deviation of eyes to right. Death in a few months after first symptoms. Lesion: a tumor the size of a walnut replaced the pineal gland, and was closely united with the choroid flexus. It was made up of numerous vessels and round cells. The ventricles were very much distended. [Nothing is said of the state of other parts of the brain, and many of the symptoms seem to point to the existence of another lesion in the left middle peduncle of the cerebellum.] Taubner.[62] Lipoma lying upon corpora quadrigemina. The case, like most cases from insane asylums, is very imperfectly reported, objective examination of fundis oculi, of sensibility, etc., being imperfect or wholly wanting. Male, aged 23 years, admitted for mania, which with remissions and periods of stupor continued until death. The pupils were at times large, at others small, always reacting very slightly or not at all (no test of vision or statement as to condition of fundus); the face and head were the seat of frequent hyperæmia. Three days before death a staggering gait was first observed. Death from pleuro-pneumonia. Lesion: a true lipoma, of the size of a hazel-nut (after hardening it measured 11 × 13 × 7 mm.) lying upon the posterior edge of the corpora quadrigemina, and upon the anterior peduncles of the cerebellum; it penetrated the subjacent nervous tissue somewhat, in the median line. It is doubtful whether any of the symptoms described in the text, with the exception of pupillary inactivity and the staggering gait, were caused by the tumor. There may have been choked disk.

(7) *Lesions of the Pituitary Body and Adjacent Region.*—Leclerc,[46] of Lyons, reports two cases of this comparatively rare affection. (1) Male, aged 64 years, not syphilitic or alcoholic, admitted to Prof. Lépine's service Dec., 1886. Seven years before had an attack of vertigo followed by loss of consciousness, without motor or speech symptoms; was well in four days. In June, 1885, severe headache, through right orbit and side of head. In June, 1886, rapid failure of vision in right eye with ptosis; also pain in left orbit and side of head, soon followed by diminished vision in this eye. In last few months much vomiting of cerebral form. Examined on admission: double exophthalmus, complete ptosis on right, incomplete on left side; double ophthalmoplegia externa; right pupil wide; almost no trace of pupillary reflex on both sides; loss of vision in right, and diminished vision on left side; atrophy of both optic nerves. [It is to be regretted that visual fields were not taken.] No peripheral paralysis or anæsthesia; anosmia shortly before death from asthenia, Feb., 1887. Autopsy: tumor size of a turkey-egg in sella turcica, replacing the pituitary body; involving the optic and motor oculi nerves. (2) Male, aged 22 years. A month before admission to hospital he suffered from pain in the chest and oppression, also neuralgic pains in the whole of left face. Soon sight failed rapidly in the left eye, and it became prominent and motionless (ptosis, etc.). No test was made of field of vision. The left optic nerve was the seat of atrophy, the right normal. No paralytic symptoms about right eye. Slight numbness of right upper extremity. Just before death there was slight paralysis of left face and of right abducens. Autopsy revealed an enormous cancer of the anterior mediastinum, with secondary deposits in various organs. Three of these were intra-cranial. Two small meningeal nodules were situated over the left motor area, at the

junction of the second and third frontal gyri with the paracentral gyrus. A much larger one was at the base destroying or replacing the pituitary body, and including the left optic tract, left optic nerve, and left motor oculi. [There must have been hemianopsia during life.] The essay is made valuable by a *résumé* of the literature of the subject.

SPINAL LOCALIZATIONS.

In the strict sense of this term, only one fact has been added to our knowledge. Westphal[35] reports the autopsy of a case of dementia paralytica in which the patellar reflex had been absent on the left side only. Sections of the cord prepared in the usual manner showed extensive sclerosis of the post. root zones at the junction of the lumbar and dorsal regions on the left side, whereas on the right side there was only a slight beginning of the sclerotic process.

APHASIA AND ALLIED STATES.

Several formal essays on aphasia have appeared during the year, one of which[2] has already been analyzed as it dealt more especially with the localization of the lesions causing aphasia. Other papers are by Arnaud, Ferrand, and Charlton Bastian. Arnaud's[65] essay on verbal deafness is an ingenious attempt to explain the various cases which are now classed under the general term of word-deafness. As a matter of cerebral physiology he admits with Munck two forms of deafness: (1) cortical deafness in which the patient hears neither words nor sounds; (2) psychic deafness in which the patient hears all sounds, but does not comprehend or understand verbal sounds or spoken words. Clinically he accepts the usual definition of word-deafness caused by lesion of the left first temporal gyrus, which he would term verbal deafness, and proposes to classify cases as follows: (1) Cases in which besides the mere verbal deafness there are lack of oral imagination, impossibility to repeat spoken words, impossibility to speak correctly (spontaneously). Such cases, which are in the majority, present paraphrasia as a prominent symptom. (2) Cases in which spontaneous speech is normal or nearly so, explicable by incomplete verbal amnesia and by recourse to visual verbal memory. (3) Cases in which the patient correctly repeats words spoken by another person, without understanding them (echolalia); he calls this mental verbal deafness. Underlying the physiology of speech in connection with word-deafness the author recognizes three distinct memories; (1) memory of oral impressions (verbal memory), (2) memory of movements of speech, (3) memory of motor oral impulses. The last he locates in Broca's convolution or motor centre for speech. The memory of movements of speech he would place in part of the general centre for muscular sense residua. The first naturally is in the left first temporal gyrus. He further admits that visual verbal memory may aid, and to a certain extent in some individuals supply the absence of the memory of oral impressions. The interconnections between these centres, and the organs of speech, and the possible breaks are illustrated by cerebral diagrams. In the second part of his essay Arnaud relates three cases of simple verbal deafness, without verbal amnesia, and with normal speech. In these cases there was more or less ordinary deafness, and the ears were not critically tested for middle and internal ear disease. This essay is marred by the use of loose terminology and some confusion is caused by the attempt at fine analysis. Farrand's[66] lectures on "The exercise of language and aphasia; anatomical lesions, and diagram" is an attempt to explain the mechanism of the various manifestations of language (spoken, written, and gestural) and their derangements according to the most recent advances in cerebral pathology. The author admits that men differ in the relative development or education of their visual and auditory centres in relation to language, and that the majority are preponderantly auditory. Also that the anatomical centres for the various functions concerned in language are active only in the left hemisphere in right-handed persons, and *vice versa.* The classification of aphasia offered is practical and sufficiently scientific, while avoiding abstract refinements. Varieties: (1) aphasia strictly speaking, or motor aphasia, (2) agraphia alone, (3) loss of gesture language alone (theoretically possible), (4) verbal deafness, (5) verbal blindness, (6) amnesic aphasia, which is further subdivided into (*a*) auditory amnesia, (*b*) visual amnesia, (*c*) kinæsthetic (motor) amnesia. The diagram or scheme which accompanies the text is well designed and serves to explain the above varieties of aphasia and possible combinations, which in practice are so common. Bastian's[67] address to the Section of Medicine of the

British Medical Association, 1887, "On different kinds of aphasia, with reference to their classification," etc., is a much more elaborate and scientific attempt in the same direction as Ferrand's, and constitutes a perfected summary of the author's previous important contribution to the same subject, tested from the standpoint of physiological psychology. Numerous memories are utilized in thought and in its expression by external signs of various sorts, and in relation to aphasia Bastian recognizes three verbal amnesias, or the impairment of three verbal memories, (1) auditory amnesia, (2) visual amnesia, (3) kinæsthetic amnesia. In health a word may be remembered, or recalled into consciousness as (1) a sound or (2) an image, or (3) a sensation (?) of certain muscular movements (motor residua) which have been used to pronounce or write it. The centre or anatomical seat of visual verbal memory is diffused in the cortex of the occipital lobe (does not attempt a smaller localization as others do,—Nothnagel[2] and others). The centre for auditory verbal memory is most likely in the posterior part of the upper (first) temporal convolution. The centre for spoken kinæsthetic memory is in Broca's convolution, and perhaps that for written kinæsthetic memory is in the posterior part of the second frontal gyrus. Admits double sets of commissural fibres between these centres. He does not admit as do Kussmaul, Charcot and others a separate, "higher" centre for verbal or ideal concepts. Prejudiced by his wholly theoretical (and opposed to many *facts*) idea, that the functions of the motor zones are not directly and truly motor, but sensori-motor or kinæsthetic, the author makes all aphasias sensory. Practically he recognizes (1) simple aphasia, (2) agraphia alone, (3) word-deafness, (4) word-blindness. Does not admit that aphasia characterized only by loss of nouns is a distinct variety. The aphasia may be due to lesion of a centre, or of commissural fibres uniting centres. The opposite scheme of classification of defects of speech and writing is worthy of reproduction, and allowance being made for the kinæsthetic theory of the author, will prove useful for the study of individual cases in practice.

The following cases of aphasia are worthy of record: Hun's[11] third case; male, aet. 66, sudden right hemiplegia with aphasia at 56, partial recovery of speech and of motion, right arm very numb and awkward. These were incomplete alexia and agraphia. He

Seat of Lesion.				*Name and Nature of Defect.*	
Cortical Lesions.	Lesions or Defects in Word Centres.	Auditory Centre.	*a.* Simple Amnesia (from Functional Defect).	1. Spontaneous Speech impaired (*Slight Simple Amnesia*).	Different Forms of Aphasic Defect in Speech or Writing.
				2. Spontaneous and Associational Speech impaired, but Imitative Speech retained, and the patient may be able to read aloud fluently (*Profound Simple Amnesia*).	
			b. Word Deafness Aphasia.	3. Word Deafness combined with complete Aphasia, owing to destruction of Centre (*Auditory Amnesia and Aphasia*).	
		Visual Centre.		4. Word-Blindness and Agraphia (*Visual Amnesia and Agraphia*).	
		Glosso-Kinæsthetic Centre.		5. Aphasia (typical) (*Kinæsthetic Amnesia*).	
		Cheiro-Kinæsthetic Centre.		6. Agraphia (*Kinæsthetic Amnesia.*)	
	Lesions or Defects in Commissures between Word Centres.	Audito-Visual Commissure.		7. *Commissural Aphasia.*	
		Visuo-Auditory Commissure.		8. *Commissural Aphasia.*	
		Audito-Kinæsthetic Commissure.		9. *Aphasia* (typical).	
		Visuo-Kinæsthetic Commissure.		10. *Agraphia.*	
Sub-Cortical Lesions.	Lesions or Defects in Internuncial Fibres.	Internuncial Fibres connecting Glosso-Kinæsthetic Centres with Bulbar Motor Centres.		11. *Aphemia* (more or less marked, difficulty in Articulation, or actual Dumbness).	
		Internuncial Fibres connecting Cheiro-Kinæsthetic Centres with Spinal Motor Centres.		No special defect known.	
Lesions in Motor Centres.	Lesions or Defects in Motor Centres.	Motor Centres concerned with Speech in Bulb.		*Anarthria* (more or less marked, difficulty in Articulation).	
		Motor Centres concerned with Writing in Spinal Cord.		No special defect (Paralysis of hand and arm).	

Classification of Some Defects of Speech and Writing.

could not spell from memory (loss of visual verbal memory). Lesion: softening of cortex of almost the whole of the post-central gyrus, the adjacent part of the superior parietal lobule, the whole of the inferior parietal lobule and the gyrus angularis. Turner[70] reports a case of traumatic aphasia in a male, aet. 20 years, who was struck by a windlass-arm on the left side of the head, causing a bowl-shaped depression, extending from about 2 inches above and behind the external angular process of the frontal bone, upward and backward almost to the sagittal suture. The greatest depression was less than half an inch. (Judging by the cut which accompanies the article the depression extended across the middle of motor zone, and its lower end was well above the Broca centre.) The symptoms were deafness of left ear, paresis of lower part of right face, right side of tongue and of right arm, complete aphasia and agraphia. Speech steadily returned, and power of writing also. At no time was there verbal deafness or alexia. When last seen there was still a trace of paresis, and there were many words which the patient could not utter. Throughout he was conscious of his mistakes and perfectly intelligent. Though observed for more than two years (1883 to 1885) no epileptiform attacks are recorded. A partial bibliography is given. Schneider,[71] of Königsberg, reports a case of traumatic aphasia cured by trephining. A man, æt. 18, received a stab-wound on the left side of the head, followed by an hour of unconsciousness, and afterward by complete absence of speech. When received in hospital four days later, the wound was found to be only about 1 cent. long and nearly healed. It was at a point 6 cent. (2¼ in.) above the external angular process extending backward nearly parallel to the sagittal suture. Symptoms: right facial paralysis, paresis of right limbs, pupils equal, complete motor aphasia. No general symptoms; understands everything, and writes his replies and desires. In the next few days complete right hemiplegia developed, and trephining was done on the ninth day. No splintering or depression of the skull was found: it was simply pierced as was also the dura. On slitting up the dura a coagulum was found resting upon the brain. On removing it a small artery spurted and was tied with catgut; the wound washed with antiseptic solution and closed with a drainage-tube. The wound healed in three or four days. On the third day after operation the patient pronounced some letters and even words; he gained words daily, though often hesitating in the choice of words; in a month speech was normal. The hemiplegia began to mend on the second day and had almost disappeared in a week. The facial paralysis was rebellious; it was still noticeable at the end of six months. The author states that this is the ninth successful case on record. A very similar case, also successfully treated by trephining, was published by Bribach, of St. Louis. Here also the injury was high up, far above the speech centre, yet aphasia was the most striking symptom, and was relieved by operation (no clot removed). Schneider's cranio-cerebral topography is externally inexact. The speech centre (Broca's gyrus) lies almost horizontally behind the external angular process of the frontal bone at a distance of 22 or 25 mm., and the seat of injury in the case is over the second frontal gyrus. Hale White[72] places on record a case of left hemiplegia with aphasia in a left-handed woman, from a large hæmorrhage in the centre of the right hemisphere destroying the knee and posterior division of the internal capsule. On the paralyzed side there was increased temperature and sweating. B. Robinson[73] presented to the New York Neurological Society a patient having ataxic aphasia without paralysis. There was agraphia, but neither cerebral deafness nor alexia. W. Briggs,[74] of St. Louis, case of cerebral concussion followed by ataxic aphasia. No depression or fracture of the skull could be detected, and there was no paralysis. At first aphasia was almost complete, and there was a degree of agraphia; no verbal deafness; reading not tested. Recovery gradually occurred. A most remarkable case of topographical amnesia, psychic blindness and word-deafness is recorded by the elder Bitot.[75] No statement as to condition of optic nerves is made, but the clinical history clearly points to psychic blindness. Smell, taste, hearing of *sounds*, and sensibility were normal; no paralysis. Aphasia was present at one stage of the illness, but was less complete at time of reporting. Unfortunately the case is not completed by autopsy. Frazer[76] presented to the Medico-Chirurgical Society of Glasgow the brain removed from a patient who had during a period of ten years presented verbal deafness and consequent aphasia; there had been also partial alexia. There was some diffused atrophy of the whole of the anterior part of the left hemisphere; there was a marked depression at the posterior extremity of the fissure of Sylvius, due to distinct atrophy of the posterior part of the superior temporal gyrus, and the posterior part of the supra-marginal gyrus; the angular gyrus also seemed slightly involved. Paget[77] reports a case in which aphasia coincided with right-sided paralysis in a left-handed clergyman, æt. 64. After another apoplectic attack there was temporary left hemiplegia without aphasia. In all attacks loss of muscular sense and severe ataxia of the arm affected (right and left) was a prominent feature. On this account chiefly is it to be regretted that an autopsy was not made. The occurrence of aphasia with right hemiplegia in this patient is very interesting, and may be explained by the fact that the patient was right-handed for writing, and had doubtless educated his left hemisphere for all the functions of language as well.

Offer[68] reports a case of possible education of the right third frontal gyrus in speech. Male, æt. 27. At 22 suffered from diplopia and vertigo. At 24 had apoplectic attack followed by right hemiplegia, hemianæsthesia and aphasia. Aphasia gradually disappeared, and the hemiplegia improved. Later paraplegia occurred. Autopsy showed caries of upper dorsal vertebræ, and transverse (pressure) myelitis. In the brain the third frontal, a part of the second, and adjacent motor gyri were found destroyed, and replaced by a cystic cicatrix. It is not stated whether patient was right or left-handed. This interesting event, recovery of speech in spite of destruction of the lower part of the motor zone, is occasionally observed in children. The reviewer has seen several such cases in which uncured right hemiplegia was present, yet the child spoke perfectly, after a varying period of aphasia. It is possible that in the adult case the patient had been ambidextrous, at least for speech.

Slow Speech from Pressure-lesion of Broca's Centre.—Robertson[78] presented to the Med-Chirurg. Society of Glasgow a tumor about the size of a marble compressing Broca's convolution in a man. When first seen the only (?) symptom was a peculiar defect in speech, consisting in great slowness and difficulty in uttering words. He remembered words quite well, knew what to say, but sometimes even after prolonged effort did not say it quite right. "The patient showed no true aphasia." The further study of the case was rendered impossible by the supervention of convulsions and unconsciousness.

Dyslexia.—Under this name Berlin[79] describes a "new" neurotic state allied to word-blindness or alexia, and bases his statement upon notes of six cases (two from other authors). The symptom (for such it is) consists in inability to read more than a few words (3 to 5) without pausing to rest, then trying again and so on. This without impairment of vision, or actual alexia, or painful sensation in eye or head. The power to read (not to see simply) is exhausted in a few moments. The author considers this a most valuable sign of beginning organic cerebral disease. All six cases ultimately proved fatal, and the autopsy showed cerebral lesions, but these were so variable in nature and location as not to throw any light on the pathology of dyslexia. In some of the cases the eyes were carefully examined and found normal. One of the above cases has been made the subject of a separate article by Nieden.[80] In this hypermetropia, one dioptric, was the only ocular fault. After death three small focal lesions were found in such locations as not to bear any relation to the dyslexia.

Stammering.—Coën,[81] of Vienna, read an essay upon a new method of treating stammering at the meeting of German Physicians and Naturalists, Berlin, 1886. The ordinary plan of treatment consists in (1) systematic inspiration and expiration exercises, (2) the exercise of the expiratory voice in gamut or scale exercises, (3) the proper and systematic employment of expiratory air for the formation of syllables, words and sentences. This is a very tedious task and requires the exercise of much care and will-power on the part of patient and physician. The improved treatment which has proved successful is based upon the observation that stammering is not present or not marked when the subject whispers. It consists of four periods: (1) a week or ten days of absolute rest for the vocal organs, no speech; (2) a week or ten days of short practice under physician's observation, of whispering speech; (3) speaking and systematic reading exercises with a slight tone, care being taken that the transition be very gradual; (4) exercises with louder voice, at different pitches, in speaking and reading. It is not so stated, but doubtless the author also includes respiratory drill and the art of utilizing the expiratory air, in this improved plan.

CEREBRAL DISEASES.

Cerebral Abscess (Suppurative Encephalitis).—The very important subject of cerebral abscess from aural disease and its surgical treatment was made the subject of a special paper by Macewen, and by a discussion in the Medico-Chirurgical Society of Glasgow,[38] on Feb'y 11th and 12th, 1887. The paper is not accessible to us, but its point was urging the desirability of treating the abscesses, cerebral or cerebellar, by trephining and drainage under antiseptic precautions. Although the author had advocated the procedure for many years he had only recently had an opportunity to operate, with a successful result. Mr. Barker, of London, referred to the various modes of extension of septic inflammatory action from the middle ear to the cerebral tissue; he claimed that cerebral abscess was three times as frequent as cerebellar abscess. The cerebral abscesses lie in a small zone whose centre is 1¼ inch above and behind the centre of the external meatus; a region corresponding usually with the junction of the squamous and petrous portion of the temporal bone, and near the roof of the tympanum, and overlying the lateral sinus. Dr. Macewen on the other hand, advocated opening the skull at a point 2 inches above the ear, and when abscess is found, to make another opening lower down. Prof. Greenfield took issue with Mr. Barker upon one important pathological question. Mr. Barker expressed the opinion that meningitis, thrombosis, pyæmia were much more frequent than abscess as a sequel of otitis. Dr. G. would claim just the opposite. Dr. Barr had collected nearly all cases[76] recorded in which abscess had been caused by ear disease, and found that 73 per cent. occurred in the cerebrum, 17 per cent. in the cerebellum, 5 per cent. in both cerebellum and cerebrum, 2 cases in the pons, and one case in the crus cerebelli. All the participants in the debate agreed as to the much greater frequency of cerebral (temporal) abscess. The symptomatology and diagnosis of abscess from meningitis and septicæmia was rather loosely treated, the drift of opinion being that high temperatures and chills were rather indicative of the latter conditions, while abscess seldom gave rise to much fever, and sometimes was accompanied by sub-normal temperatures. Such symptoms as headache, increasing stupor to coma, and local tenderness to percussion were thought the most reliable signs of abscess; besides a slow, and not rarely remittent course as compared with meningitis. Localizing symptoms strictly speaking were admitted to be wanting. What may prove, after verification, to be a symptom of abscess, is the condition of the urine. W. J. Somerville[49] had analyzed the urine in two cases, and found the chlorides far below the normal, while the phosphates were much increased in quantity,—suggesting excessive metamorphosis of brain tissue. In one case, successfully operated by Macewen, the urine gradually returned to its normal state after the operation. As cases of supposed cerebral abscess from aural disease are nearly always emergency cases, requiring prompt decision and operation, this new aid to diagnosis will be seldom applicable. With respect to optic neuritis, most of the members supported Dr. Greenfield as against Gowers and Barker, that it is very rare in cerebral abscess. The drift of the whole discussion was to establish on a scientific foundation the doctrine of freely opening and cleansing cerebral or cerebellar abscesses from ear disease at a not too late period, before full coma sets in. At the same time the middle ear and mastoid cells should be thoroughly explored, freed from pus and necrosed bone, and well drained. The results of operations thus far performed is most encouraging, and lead to the hope that many lives will henceforth be saved by this procedure. Köhler[39] successfully trephined for an extra-dural abscess due to otitis media. He was guided by slight oozing of pus 4 cent. directly behind the external auditory meatus. In this case we find corroboration of the diagnostic rules advanced in Glasgow, viz., in Köhler's case there was very decided pyrexia at the beginning, the temperature ranging from 38° C. to 40.5° C., and the lesion was not strictly speaking cerebral abscess.

The above abstract refers to the semeiology and diagnosis of cerebral abscess from aural disease. In other kinds of cerebral abscess the diagnosis turns largely upon knowledge of the antecedent pathological cause, such as cranial injury, caries of ethmoid or orbital bony regions, empyema, phthisis, pyæmia, etc., together with the few brain symptoms, and as a rule with little or no pyrexia. Westmoreland[84] speaking of traumatic abscess, expresses the same opinion; a normal or subnormal temperature is generally present, whereas fever is the rule in cerebral affections which may be mistaken for abscess. Idiopathic cerebral abscess is we believe impossible to diagnosticate. The rapid wasting referred to by Prof. Greenfield[38] and the peculiar condition of the urine[49] may here prove valuable aid.

Pathology.—In the above general discussion Greenfield stated that the majority of abscesses were due to septic absorption, usually accompanied by septic phlebitis. The dura was first involved, the brain becomes adherent, and a local gangrene of the brain results, spreading from the veins first affected. Workman[38] (and Macewen in his paper) stated it as a fact that cerebral abscess never occurs without some septic germ having previously gained admission. Coats called attention to the almost invariable presence of putrid or gangrenous odor in cerebral abscesses. Microscopic examination of the contents of an abscess showed (by Gram's staining) numerous (perhaps twelve) forms of cocci, short rods, long rods, etc. In a section of the surrounding membrane very few isolated organisms were visible. He considers the cerebral lesion to be due to advance of putrescence and necrosis from the ear, rather than to a true infection by a special coccus. In commenting upon his case of cerebral abscess following empyema, Drummond[7] expresses the opinion that it was due to an embolic process. A. Fraenkel[82] in an elaborate paper on tubercular cerebral abscess, expresses himself as follows: The lesion (tumor or abscess) was clearly indicated by paralysis of the right arm and face, deviation of the tongue, motor aphasia and great tenderness to percussion over the left central gyri, no spasms. An abscess was found in this region, but wholly in the white substance; its contents odorless. On microscopic examination the organized portion of the cyst was found free from tubercle bacilli. These were abundant in the true granulating layer of the cyst, and still more so in its fluid contents. In the granulation tissue the bacilli lay between the young cells. He believes that the abscess originated in the breaking down or liquefaction of a large conglomerate tubercle of the brain.

Drummond[7] and Delbet[23] report cases of cerebral abscess (probably embolic) from encysted empyema. An abstract of Drummond's case will be found in the paragraph on the facial centre. In Delbet's case, although their diagnosis was reasonably clear, no operation was attempted and the patient allowed to die without treatment.

Treatment.—As indicated in the discussion in the Medico-Chirurg. Society of Glasgow[38] (*supra*), a vast advance has been made in the treatment of cerebral abscess, and henceforward many lives will doubtless be saved by operation. In the reports of recent operations for the relief of abscess in the brain or cerebellum one is very strongly impressed with the fact that in many cases the operation was delayed much too long. In the present state of our knowledge, with the evidence in our possession of the relative harmlessness of exploratory trephining of the skull, the operation should be done just as early as a medical diagnosis of the case can be made. In a few cases of pyæmic or embolic abscess (*e.g.*[82]) the diagnosis may be made early by rules of localization; and in such a case, if there be a doubt as to whether the lesion be a tumor or an abscess, the operation should be attempted. In surgical cases the location and extent of the injury are guides to the finding of the abscess. In abscesses from aural disease, which usually lie in the temporal lobe, but which may also be in the lateral lobe of the cerebellum, opinions as to surgical procedure vary. For cerebellar abscess the plan of operation is clearly indicated. For abscess of the temporal lobe Macewen advises trephining at a point two inches above the bony meatus, Barker at a point 1¼ behind and above the meatus, while McBride[83] states that the best place for opening the skull is just in front of and above the external meatus. In all these operations the measurements are from the centre of the bony meatus. Studied upon the skull, both Macewen's and Barker's opening are probably well suited to find pus in the temporal lobe; perhaps the latter is best. But McBride's opening just in front of and above the meatus would give access to the anterior surface of the petrous bone too far forward to reach the points where necrosis and meningitis usually occur. Westmoreland[84] reports a case of chronic (7 years) traumatic abscess cured by operation. Link[85] describes an unsuccessful case (operation postponed too long) complicated with suppurative lepto-meningitis. There was sharp fever in the last few days of life.

CEREBRAL HÆMORRHAGE.

In the way of *semeiology* and *diagnosis* of this affection nothing new has been published. Relating to difficulty of diagnosis, the following case by Hodge[89] is perhaps worthy of mention.

Female, aet. 19 years; supposed scarlet fever, history of acute rheumatic attacks, valvular disease of the heart recognized. Apoplexy following headache and rigor, accompanied by left hemiplegia. No temperature given. Death in twenty-one hours. Lesions: vegetation on mitral and aortic valves; extensive cerebral hæmorrhage in right hemisphere, bursting into ventricles. State of vessels not mentioned. The patient's age and the presence of valvular disease made a diagnosis of embolism almost a necessity.

Pathology.—The general subject of diseases of the cerebral arteries as a pathological basis for a great variety of cerebral lesions, is admirably treated by Gerhardt.[86] He briefly studies all forms of disease of the arterial system of the encephalon, and furnishes many useful anatomical and physiological data (size of vessels, pressure in skull, etc.) Cerebral hæmorrhage is usually caused by miliary aneurisms, which are the result of endarteritis (Bouchard is not mentioned); but it may also be the result of scorbutus, leucocythæmia, pernicious anæmia, etc., and of excessive strain increasing intra-cranial pressure (pertussis). A more critical and anatomical study of miliary aneurisms is by L. Lowenfeld.[87] The initial lesion is in the endothelial layer of the small arteries, involving also the membrana fenestrata, and causing fatty and granular degeneration of the muscularis. After this lesion is developed, the artery is ready for dilatation, usually in a globular form, though sometimes in ovoid or elongated dilatations, and dissecting aneurisms. Although this is unquestionably the lesion causing the majority of spontaneous cerebral hæmorrhages, yet there are other causes, such as simple or fatty atrophy of the vascular walls; lesions of veins in some cases. The causes of cerebral hæmorrhage (apart from lesions of arteries and veins), are classified as follow: (1) Increased arterial pressure from cardiac over-action, from contracted kidneys, arterio-sclerosis, constipation, emphysema, abdominal fat, venous thrombosis in cranium, etc. (2) Increase in the total quantity of blood (plethora). (3) Changes in the blood itself, such as various intoxications, anæmia, syphilis, rheumatism (?) and gout. He is inclined to allow a prominent part in the mechanism of hæmorrhage to psychic and reflex nervous disturbances. Inherited disposition to cerebral hæmorrhage he explains by inheritance of deficient development, or resistance-power of cerebral blood-vessels. There is apparently no advance in these views over those published in 1868 by Bouchard and Charcot. Though often misrepresented, these authors did not claim that *all* cases of cerebral hæmorrhage were due to the lesion which they established as the usual one, viz., miliary aneurism; but they stated that thrombosis, anæmia, etc., in certain cases were the lesions causing the hæmorrhage.

Under the title of "apoplectiform pernicious paroxysm, with and from cerebral hæmorrhage," Blanc,[90] a French army surgeon stationed in Tunis, makes quite a thorough study of the subject of the double relation of cerebral hæmorrhage and pernicious intermittent fever. The paper is based upon a case in which after several quotidian paroxysms during the prevalence of congestive fever in Tunis, one night epistaxis occurred, followed in the morning by convulsions and apoplectic state. Stertorous breathing, pupils contracted, pulse 90, temp. 38°.7 C. Death in 2 hours. The subject was a strong, healthy man of 23. Autopsy showed a very large hæmorrhage in the left hemisphere, in the centrum ovale, bursting through the cortex, and extending under the arachnoid. In the ragged walls of the focus were numerous dark pigmented and dilated blood-vessels. In various places sections reveal miliary hæmorrhages. The cortex generally was intensely congested. No technical search for aneurisms appears to have been made. Some of the author's conclusions are (1) The pernicious apoplectiform paroxysm is almost always preceded by intermittent paroxysms of the ordinary kind. (2) It may be preceded by hæmorrhages in various parts. (3) It may be accompanied and followed by various paralyses, or even by hemiplegia. When permanent these palsies are probably due to cerebral hæmorrhage; when transitory they may depend upon intense congestion or temporary thrombosis. (4) The temperature is always moderate in malarial apoplexy, and never, on the one hand, attains the height it does in ordinary paroxysms, nor, on the other hand, does it ever fall as low as in the early stage of common cerebral hæmorrhage. (6) Malarial congestions tend in all viscera to a sub-acute or acute inflammatory state, or they may give rise to minute or large hæmorrhagic infarctions. Melanæmia, or melanosis of various organs are reliable signs of malarial intoxications.

Treatment.—L. Braddon[88] in a pretentious article, advocates the surgical and mechanical treatment of spontaneous cerebral hæmorrhage; advocating compression of one or both carotids, opening the skull to relieve pressure, and even opening the hæmorrhagic focus and removing the clot. This rash suggestion is based on a one-sided view of the lesions of cerebral hæmorrhage and on a misplaced confidence in our ability to make a diagnosis of hæmorrhage. "So certain is the diagnosis," etc., is a phrase which embodies the author's delusion on this point. In reality, in most cases the diagnosis between hæmorrhage and thrombosis with consequent softening (and in many cases embolism and consequent softening) is extremely uncertain. Perhaps the only reliable guide is the temperature curve, and in many cases the physician does not see the patient soon enough to obtain the initial lowerings of temperature which Charcot and others consider as characteristic. The author is, furthermore, absorbed in the idea that a clot does most damage by pressure. In reality it is the laceration of important gray or white substances which causes the irreparable mischief, and removal of the clot is useless to repair it. Very probably also a sudden change in the intracranial pressure by trephining, and, more sure still, the removal of the blood would favor the escape of more blood from the ruptured aneurism, or cause rupture of other miliary, ectatic, or dissecting aneurisms. The clot, within certain limits, is, in our opinion, a help to the arrest of hæmorrhage, and it is well known that it is quickly and readily absorbed. If no important centres or fasciculi have been torn by the hæmorrhage in the first phase of the attack, recovery is perfect. The suggestion to compress the carotid on the side of the lesion, is also, we believe, dangerous. Contrary to what seems the author's idea, the bulk of the effusion is thrown out at once, before the physician can see the sufferer; we are absolutely without proof that (in the majority of cases) bleeding goes on for any length of time. Besides, the cerebral tissues round about the clot, and in many cases the whole brain, is anæmic immediately after the hæmorrhage, from pressure, and this is certainly a contra-indication to interfering with arterial supply, as it is to copious phlebotomy. We have perhaps given too much space to this criticism; but as a "craze" is threatened in the way of trephining the skull for all sorts of lesions we wish to do our share in attempts to restrict the application of this most valuable operation to conditions and cases where there is a rational and promising indication for its performance.

INTERMENINGEAL HÆMORRHAGE.

A case of this sort, secondary to a scalp wound, successfully treated by trephining, is reported by Armstrong.[126] The patient, a male adult, had received a blow upon the left side of the head, tearing the scalp, but not injuring the bone. A month later right hemiplegia gradually developed. After trephining over the left motor area, the large needle of a hypodermic syringe was introduced through the dura-mater and gave issue to several ounces of disorganized blood. Carbolized horse hairs were introduced through the enlarged opening. Four hours after the operation, the patient was able to move the paralyzed extremities, and convalescence occurred without local reaction or sepsis. When discharged, his muscular power was as good as ever. It is to be regretted that the inner surface of the dura was not examined for traces of pachymeningitis. The late appearance of symptoms would take the case out of the ordinary category of traumatic meningitis. The operation should be made more thorough by raising a flap of the dura after using a two inch trephine (Horsley's method). In many cases hæmorrhage occurs on both sides of the head, or altogether on the side opposite the cranial injury by *contre coup*. In such cases, according to indications, we think that the trephine should be used on one side or on both sides, and the intermeningeal space cleared of clots of blood as far as possible. The theoretically good prospects of this operation are reduced by the fact that in a certain (or rather an uncertain) proportion of such cases there is laceration of brain substance.

CEREBRAL THROMBOSIS.

Semeiology.—Case by Dr. Hanford.[93] Boy, æt. 5 years. Headaches, constipation, frequent vomiting. No aural disease. Death in coma about ten days after beginning. The autopsy showed adherent decolorized clots in the superior longitudinal and lateral sinuses. The superficial cerebral veins were also thrombosed and felt like whipcords. Numerous hæmorrhages on the surface of both hemispheres, some ploughing up the brain substance considerably. Hæmorrhage evidently recent and its derivation uncertain. Diagnosis impossible. Chafrely[94] presented specimens of this disease to the Société Anatomique de Paris, due to a disease of the eyelid (?).

HÆMORRHAGIC PACHYMENINGITIS.

Semeiology and Diagnosis.—Grainger Stewart[91] reports the following traumatic case. Male, æt. 44. Fall on back of head, causing momentary loss of consciousness. In a fortnight headache (frontal pain), with giddiness, weak legs, and, later, staggering. On admission to hospital, no outward sign of injury to head, no actual paralysis, no anæsthesia; vision good, but double optic neuritis present; occasional vomiting and torpor; temperature subnormal (97° and 97.8°). Eight weeks after injury almost comatose, well marked aphasia, right hemiparesis with anæsthesia. Trephining was done by Mr. Annandale over the left third frontal gyrus. On opening dura some six ounces of red-brown fluid blood escaped. Marked improvement followed. Fifty hours after operation, headache, rigor and spasm began. Later, convulsions in all extremities, more on right side, increased fever, coma. Death on sixth day. Autopsy revealed pachymeningitis interna, with blood and clots on both hemispheres; also purulent leptomeningitis (result of operation ?). Clemow[92] records an idiopathic case well illustrating the difficulties of diagnosis. Female, æt. 67. Severe headache, chiefly frontal, worse toward evening; anorexia and nausea. Headache had lasted eight or more weeks. Pupil and pulse normal (60 ?). A few days later drowsiness and more nausea; very severe headache. A week after first examination a general convulsion; speech less distinct, more drowsiness; weaker. Both optic disks hyperæmic. Slight twitching of muscles of face and arms. No actual paralysis or anæsthesia. Pulse rising (85). On seventeenth day patient brighter and apparently better, but this soon passed into wakefulness with delirium. Temperature had been low, 97.6° to 98.2°. On twenty-fifth day died comatose, after variable periods of stupor, momentary clearness of mind and speech, general hyperæsthesia. State of pupils and optic nerves not again mentioned, and final temperature not given. Autopsy showed hæmorrhagic pachymeningitis over both hemispheres, the clot being from $\frac{1}{10}$ to $\frac{1}{8}$ inch thick.

Treatment.—The difficulty of diagnosis is so great that it might be said there is no recognized treatment for this disease. In case of probable injury, we should favor a trial of the operative treatment, trephining on both sides, washing out the clot as far as possible, and administering very large doses (from 100 to 500 grains a day) of iodide of potassium. More especially in traumatic cases this might prove successful. The cortex in them is injured only by pressure, and iodide of potassium is well known to exert a peculiarly curative effect on pachymeningitis generally.

TUBERCULAR MENINGITIS.

Semeiology.—A new symptom is claimed by Skeer,[95] of Chicago. If verified, it will certainly be welcome,—as much uncertainty yet exists in the diagnosis of this affection. "The symptom to which I allude is a small circle which forms in the iris, near to and completely surrounding the pupillary margin. When it begins to appear it is very indistinct, and resembles a wreath of thin white clouds, the edge of which extends at first to the free border of the iris. In from 12 to 36 hours, the whole margin of the iris will be involved, having become of a whitish or yellowish-brown color, and appearing irregular, thickened and somewhat granulated." The cloudiness is very evanescent and should be looked for at each visit; it is best seen in brown irides. The author has observed these wreaths in twenty or more cases, in two of which the diagnosis of tubercular meningitis was verified. "When the wreaths of white clouds appear in the iris in a case of cerebral meningitis, I consider the question of diagnosis settled beyond a doubt." A committee of the Chicago Pathological Society was placed in charge of this question, and by correspondence with various oculists and physicians, ascertained that the appearance had never been observed. No one has as yet verified Dr. Skeer's statements.

Jules Simon[96] has published a valuable lecture on the differential diagnosis of tubercular meningitis from several diseases, viz.: typhoid fever, pneumonia (in which violent cerebral congestion may appear), early stage of eruptive fevers, intermittent fever; congestive attacks in the course of infantile cerebral sclerosis, school-headaches with vomiting, true migraine, and lastly cerebral symptoms of reflex origin (from otitis, teething, worms, etc.). This lecture is not suited for analysis, but should be read. An American translation will be found in Ref. [97]. Lépine[99] observed a case in which almost the only symptom of a tubercular meningitis was hemiplegia. Male, æt. 29 years, having had pleurisy two years before. First symptoms were dragging of the left leg and formication in whole of left side of body, with some involuntary twitchings of the left fingers. There was some headache and slight hesitancy in speech. Soon the hemiplegia became complete, and the right side showed some paresis; vomiting, insomnia, delirium; very slight fever. Autopsy showed meningitis with discrete tubercles. One patch lay on the right paracentral lobule. The lesions at the base were trifling. There were some pulmonary tubercles and the remains of the old pleurisy. Belfield,[103] of Chicago, has published a case simulating opium poisoning (compare Wilkes[104]). Girl, æt. 20 years, of a non-tubercular family. Five days before death headache with vomiting occurred. Was better after this till next day, when a similar paroxysm occurred, and again on the third day. The pain was extreme. In the night she became comatose, with slow stertorous breathing (as slow as 4 to 5 per minute), contracted pupils, with a suggestion of strabismus, warm skin (no temperature given), pulse 80. Autopsy revealed enlarged and cheesy glands in the abdomen; the membranes of the brain were closely adherent to the brain substance and abundantly studded with miliary tubercles. F. Schulze[105] records three cases which during life presented such symptoms that the only possible diagnosis was acute meningitis, yet at the autopsy the membranes were found normal. There were lesions, however, in the cerebral and spinal substance, consisting of intense hyperæmia and accumulation of round cells about the blood-vessels. In the discussion Leyden, stated that it was possible that the lesions of meningitis had existed in Schulze's cases and disappeared before death.

A remarkable case of reflex cerebral symptoms simulating meningitis, is reported by Devaux.[106] A healthy girl of three years, who had never had convulsions; dentition easy. For a few days before appearance of cerebral symptoms had a "cold," was often tired, and awoke weeping in the night. In the afternoon, after a fatiguing day, chilly sensations, nausea and general convulsions, both tonic and clonic, more marked on the left side; stiffness of neck, trismus and grinding of teeth, conjugate deviation of face and eyes to left. The pupils were normal. Pulse irregular at about 140, respiration very irregular (at times of Cheyne-Stokes type), temperature 38.3°. There was also a peculiar, rhythmic, nervous cough which continued to the end of the brain-symptoms. The convulsions recurred frequently during the night. Next morning 10 grains of calomel were given. More or less convulsive action continued throughout the day; the abdomen was concave; there were a few sibilant rales in chest; pulse 120. Temperature 38.5°. No vomiting or pupillary symptoms. In the early afternoon three movements occurred, bringing away an enormous quantity of oxyuris. Nervous symptoms and fever subsided at once. On third day 7 grains of calomel were given; very well all day; in the night, return of convulsions with intervals of stupor. Next morning well again. Ordered 10 grains more of calomel. Remained well. In this case although the absence of prodromata, of tubercular inheritance, of vomiting and pupillary symptoms was against meningitis, yet the physician was well justified in feeling a doubt as to the diagnosis, considering how protean tubercular meningitis is. The author advises giving large purgative doses of calomel in all such uncertain cases.

Kahler[114] confirms the statements of Rendu, Schulze and Reymond, as to the extension of disease to the nerve roots in tubercular meningitis. Among the unusual symptoms were ptosis and paralysis of the entire oculo-motorius on the right side, twitching of the facial muscles (and of those of the limbs also), left-sided facial paresis, paralysis of the right sixth nerve. The optic nerves were normal. Autopsy showed a tubercular gelatinous exudation at the base of the brain; general miliary tuberculosis; ancient tubercular foci in the apices of both lungs and in bronchial glands. Microscopic examination showed a nearly normal left oculo-motorius, while the right was extensively diseased by cellular infiltration of the perineurium penetrating into the nerve, and dilatation of the vessels. Numerous fibres were degenerated, others showed uncolored myelin (Weigert method), and there were rounded light spaces of various sizes, apparently filled up with coagulated material, and showing here and there an endothelial lining. These spaces the author considers as belonging to the lymphatic system, and communicating with the sub-arachnoid lymph-channels, whence had come the exudation. (A somewhat similar process has been described as affecting the optic and the acoustic nerves, in tubercular and in cerebro-spinal meningitis.)

Prognosis.—Simon[96] expresses his absolute disbelief in the curability of tubercular meningitis, but other authors do not share his views. Carrington[98] presented to the Pathological Society of London, specimens removed from a lad 16 years of age. After injury to one knee, both knees became inflamed, later psoas abscess appeared on the right side, after which he improved. A few months later, a psoas abscess of the left side caused death. Among the various lesions found were those of cerebral and spinal tuberculosis. In the cerebral membranes small yellow, caseous tubercles were found, and the microscope verified their tubercular nature. No symptoms had betrayed these lesions during life, so that the author's claim is hardly justified; the case was rather one of latent cerebral tuberculosis. (*Vide* treatment.) Lugeol,[107] of Bordeaux, related to the medical society of that city a case which appeared to him to be tubercular meningitis ending in recovery.

Treatment.—Some time in 1886, Dr. Warfvinge, of Stockholm, reported five successful cases treated by inunction of iodoform to the shaven scalp. One gramme (gr. xv) of iodoform mixed with five grammes of vaseline, was rubbed in night and morning, and the head kept covered with an oiled silk cap. In Dec., 1886, a discussion[100] upon this new method was held in the Medical Society of Stockholm. Warfvinge repeated his statement. Medin had tried the treatment in eight cases, all of which proved fatal, and out of which seven autopsies showed a correct diagnosis. Six other cases treated by other methods also died. Waern had failed with iodoform in several cases; one case of cerebro-spinal meningitis recovered. Lindblad reported eight cases, of which seven proved fatal. The general opinion seemed to be that the reported good results were in cases which had not been tubercular meningitis.

Martel[101] reports three successful cases in which the same treatment was carried out for many days. The cases were of meningitis, but doubt must remain as to their tubercular nature. The same plan of treatment has been tried by R. C. Holt[102] in five cases, of which two died. The diagnosis in all the cases is very questionable. Lardier[108] relates a case which he believes illustrates the utility of tannin in the treatment of non-tubercular meningitis, but the diagnosis of his case is more than doubtful; its course and many of the symptoms point to pneumonia with associated cerebral congestion. At the first visit "a little pulmonary congestion" at the base of both lungs is noted, but these organs are not again mentioned. Ice to the head and tannin, 3 grains every three hours, constituted the treatment. A defervescence followed the day after, and no further rise took place. The temperature curve was that of pneumonia.

CEREBRO-SPINAL MENINGITIS.

Semeiology and Diagnosis.—J. Lewis Smith[115] read a paper upon this affection, or as he calls it, cerebro-spinal fever, before the Northwestern Medical and Surgical Society of New York, in which he dwelt more especially upon some of its anomalous symptoms, etiology, and its rapid spread through this country. Only a few of these points can be presented here. First, he shows, from Health Board Reports of New York City for 1883 and 1886, that the disease prevails about equally at all seasons. Second, that infants under one year furnish the largest number of fatal cases, and that nearly nine-tenths of the fatal cases (in 1883) were in patients under 21 years of age. Third, he shows that the disease is prevalent, and probably endemic, in all the larger cities of this country, and especially in the Northern States. Fourth, in many cases the onset is sudden and proves rapidly fatal; and the suggestion is thrown out that some cases diagnosticated as infantile cerebral paralysis or polioencephalitis, are in reality cases of cerebro-spinal fever of great acuteness, and resulting in hemiplegia, with or without aphasia. Fifth, the disease is usually primarily cerebro-spinal in its manifestations; but it may also occur as secondary to pneumonia and typhoid fever. In "many cases" hemiplegia has developed on the second day. In the discussion which followed, Peck[115] referred to·the aural and optic complications. In the former case he holds that the pus travels along the acoustic nerve sheath to the internal ear. He had examined the eyes in many cases of various forms of meningitis, and often found perioptic œdema, succeeding to infiltration of the optic nerve sheath; some had loss of central vision. "Almost all cases of inflammatory œdema of the basal meninges are followed by neuritis descendens and neuritis optica." Levick,[116] of Philadelphia, writes a commentary upon Dr. Smith's paper, in which he claims (contrary to Smith) that the disease was known in the last century and even as far back as the sixteenth century. He thinks that the influence of the germs of the disease shows itself in masking the symptoms at onset of other diseases (severe headache, cervical pain and stiffness, cutaneous hyperæsthesia at beginning of typhoid fever). He considers epidemic catarrhal fever and cerebro-spinal fever as closely allied. Davidson[117] reports a most remarkable case of sporadic nature. Male, aet. 30, sudden onset, with pain in head and limbs, followed by opisthotonos, moderate fever (never over 101°), delirium, petechiæ and measles-like eruption. In about 12 days, patient passed into a condition of "purely organic existence, without any trace of rational power," which lasted 75 days and terminated suddenly. In this period the patient did many complicated acts fairly well (washing his face, attending to wants, making movements of his occupation, etc.), and acting deliriously on several occasions. Speech suspended, and apparently he understood nothing. Marked anæsthesia of extremities. Speech and intelligence gradually became normal. Towards the end of convalescence several epileptic attacks occurred. The treatment consisted in repeated blisters to nape of neck, mercury and iodide of potassium internally. Morphia and bromidia were given for sleeplessness and maniacal outbursts. The possible relationship, at least in an epidemiological sense, between meningitis epidemica and typhoid fever, is strongly argued by Steiner,[118] from his study of epidemics of the true disease in the town of Rosenberg, one overlapping the other, the typhoid cases having occurred first. The surrounding districts did not suffer.

An interesting monographic report of an epidemic of cerebro-spinal fever is that of Hermann and Kober,[119] of Beuthen, Germany. It would prove of great advantage to medical science, or at least of medical hygiene, if physicians in small cities and towns were to follow this excellent model of a report in this country. The origin of the epidemic they trace to a transmission of the virus through occasional sporadic cases since the epidemic of 1877. The disease was not localized in any one part of the town, but it showed a strong preference for certain houses. The authors consider the virus as a locally produced one. They deny its contagiousness and rest this assertion largely on the fact that no new case developed in the hospital which received many of the patients. In all 85 cases were recorded by the two observers.

33 per cent. proved fatal. As regards age of subjects, their results conflict with those of American physicians. Only three subjects were under one year of age, nineteen from one to ten years, and nineteen from ten to twenty years. The sexes were about equally affected. Prodromata, debility, anorexia, nausea, slight fever, were common; and the disease began with severe headache, vomiting, staring look, followed by cervico-dorsal pain, increased by pressure upon the spinous processes and by passive movements; opisthotonos and irritability. A wholly irregular temperature curve was the rule. Remissions frequent, and often led to gradual recovery. Often death ensued, with more violent head-symptoms, tetanic spasms, convulsions, coma and paralysis of the respiratory centre. Many exceptions to the above regular semeiology occurred; often sudden onset was followed by epileptiform or apoplectiform states. The symptoms are minutely analyzed in the original, and we note the following only. In a few cases hæmorrhagic nephritis, with enormous quantity of blood in the urine, and at the same time large cutaneous hæmorrhages. Occasionally polyuria, with and without sugar. Hemiplegia was observed only once; temporary blindness several times; deafness twice. The deep and superficial reflexes were usually irregular. Herpes (naso-labialis) occurred in fully one-third of the cases. Erythema, roseola, were rare, petechiæ frequent, and in only a few cases was articular exudation observed. Ten cases were examined post-mortem. In four cases, besides purulent infiltration of the pia, there was purulent exudation outside of it (in intermeningeal space). In three cases the lateral ventricular walls were covered with a thin purulent layer. The same epidemic was also studied by S. Richter.[120] He observed 56 cases, with a mortality of 48 per cent. The author is inclined to look upon the disease as miasmatic and contagious. According to Netter,[122] pulmonary meningitis may present itself in four symptom-groups: (1) Wholly latent and revealed only by autopsy. (2) With symptoms of cerebral excitation grouped so as to form the classic picture of convexity meningitis. (3) An apoplectic form, very rapid development, or even a truly apoplectic one, with hemiplegia in some cases. (4) Cases in which cerebral symptoms occur at the same time as or even earlier than the pulmonary ones. Wilcox[123] reports four cases in which distinctly meningitic symptoms were

complicated later by pneumonic ones. All of these cases (children from 8 to 13 years old) recovered. Zitéke,[124] of Batesville, Indiana, writes an interesting paper on the disease, giving quotations from old writers (16th century), etc., and describing an epidemic he witnessed in 1879. Nine cases occurred in one school. Leyden[125] considers it as proven that the pneumococcus pneumoniæ causes cerebro-spinal meningitis as well. In the lungs the coccus can be discharged by expectoration, in pleurisy it is thrown out with the effusion, but in the brain and spinal cord it is confined and can only die *in situ* by disintegration. He finds a strong corroboration of this view in the fact that in autopsies he has made of the disease, at a late period, the pneumococcus could not be found. Relapses or exacerbation he attributes to fresh growths of the microbe. The greatest danger in meningitis lies, not in the virulence of the infection, but in the local injury done to the brain on its surface or in the ventricles. Another source of danger is the extreme emaciation which occurs in prolonged cases, and which Klebs has compared in degree to that of phthisis and carcinomatous disease. Consequently vomiting is one of the most actively dangerous of the symptoms by favoring starvation (and by the straining effort), and this establishes the indication, which he thinks is the prime one, to check this symptom by morphia.

Etiology and Pathology.—Smith[115] quotes extensively the observers (Fränkel, Eberth, Pashkareff and others), who have lately claimed that the disease is caused by a micrococcus, similar to or identical with the coccus of pneumonia. He does not consider the identity as proven, and thinks that this subject needs much more investigation. Smith "does not believe that the disease is contagious at any time." Weichselbaum[121] has made numerous experiments with the cultures of the coccus of pneumonia and meningitis. Mice, guinea pigs, rabbits and dogs received injections of cultures of the diplococcus intra-cellularis meningitis, in the pleura, abdomen and intermeningeal space. Acute inflammation resulted in all cases, with reproduction of the diplococcus. The cerebral experiments in dogs were particularly interesting. Not only acute pachymeningitis and leptomeningitis were produced, but also encephalitis; also effusion, more or less purulent and bloody, in the ventricles. In one of these three dogs, strange to say, neither the cerebral abscess nor the meningeal pus contained cocci, or gave any result of culture. The occurrence of encephalitis with simple red softening or abscess in these cases is particularly interesting in connection with Smith's opinion (vide p. 53) that polioencephalitis may be due to cerebro-spinal fever. The author concludes that both the pneumococcus (Fränkel, Klebs, Leyden, Eberth and others), and the diplococcus intracellularis meningitis may be causes of cerebro-spinal meningitis with or without pneumonia. The modes of entrance in cases of meningitis he considers to be frequently the nasal passages (as advocated by Strümpell and Weigert), especially if, as often (?) is the case, a sharp nasal catarrh precedes the cerebral symptoms. The cocci may also gain entrance to the brain through a diseased tympanic cavity. (Vide p. 53 Levick's opinion of the relationship between epidemic catarrh and cerebro-spinal fever.) Netter[122] has published two parts of an exhaustive clinical and experimental essay on the pathology of pulmonary meningitis, and reaches the conclusion that this disease, at times so prevalent and so fatal, is caused by the pneumococcus. His results as to meningitis without pneumonia have not yet been published. Wilcox[123] adds to his clinic notes a summary of researches and opinions bearing upon the relations between the pneumococcus, meningitis and pneumonia. The same microbe has been demonstrated in malignant endocarditis and its complications, by Netter.[122]

Treatment.—Smith[115] and Levick[116] say little on this subject, or at least nothing new. Herman and Kober[119] discuss the prophylaxis and treatment of the disease quite fully. They recommend, when a case occurs in a dwelling, that it be instantly vacated, the sick being removed to a hospital, and the place thoroughly disinfected and left uninhabitated for several weeks. They also recommend the usual sanitary police regulations as to overcrowding, ventilation, cleansing of houses, etc. The treatment should be largely an energetic antiphlogistic one; ice to the head and spine, and in strong persons, leeching or wet-cupping. Purging with calomel or jalap; bromide of sodium and chloral for excitement; morphia only seldom and for severe pains. A tonic diet and, under some indications, wine is required. Zitéke[124] (Jan., 1887) employed morphia hypodermatically, with simple rectal alimentation of Carnrick's beef peptonoid, in one case, with rapid and thorough success. One-sixth grain of morphia sulphate was thus given frequently (almost every hour) on the first day of treatment, and less often afterward, and omitted on the ninth day. Each injection was followed by cessation of irritative symptoms and rest; the vomiting was also controlled. Leyden[125] considers rest and avoidance of painful movements of the neck of the greatest importance; constant feeding with small quantities of strong liquid food, such as milk, iced tea, albuminous preparations (eggs and gelatin), peptonized milk, and relatively large quantities of brandy or sherry. As to medicines, he is indifferent as to the use of mercury, without denying that it has seemed to do good occasionally. Antipyretics he considers as useless, if not injurious. Iodide of potassium cannot be expected to produce any specific effect. He is opposed to baths chiefly because their administration requires more or less movement of the patient's body. The mainstay for him is morphia or opium, administered with the double object of controlling vomiting and keeping the patient comfortable and quiet. He makes no mention of counter-irritation to the spine or neck. A most interesting case is reported, with temperature curve, ending in recovery. The initial or prodromal period lasted nearly three weeks, the stage of full development of symptoms two weeks, and the stage of decrease nearly three weeks more. In this stage the patient very nearly died of collapse (temp. 36.4° and 36.2°) and was saved only by the utmost exertion. In this case muriate of morphia was principally used, given hypodermatically in doses of 0.01 ($\frac{1}{6}$ grain) every three, four or five hours, according to vomiting and distress. Leyden also recommends this plan of treatment (absolute rest, morphia injections, small quantities of strong food given frequently per os and per rectum) in the vomiting of acute infectious diseases in general.

CHRONIC MENINGITIS.

Bruce[109] showed to the Medico-Chirurgical Society of Edinburgh a brain with the following lesions: Convexity normal; pia, at the base, especially over pons, sides of medulla and cerebellum, milky and thickened. The lateral, the third and fourth ventricles were dilated; the ependyma granular; foramen of Monro half an inch in diameter. Venæ Galeni not obstructed. The specimen was removed from a girl under the care of Dr. Carmichael, who had diagnosticated simple meningitis. After having been dismissed as "cured," she died in general convulsions. The author ascribes the hydrocephalus and general convulsions to the glueing of the cerebellum and medulla, and consequent obliteration of the foramen of Magendi. Klippel[110] reports a case of chronic meningo-encephalitis limited to one motor area. Female, æt. 42, no tuberculosis. In September, 1886, began to have unilateral convulsions beginning with an aura of pain in the right hand and arm; the arm was first convulsed, then the leg, and the face and eyes turned toward the right; loss of consciousness; no biting of tongue. These attacks recurred until death in February, 1887. On the last day she passed into status epilepticus, having 56 seizures from noon to 5 A.M.. The spasms affected both sides at the last (tonic-clonic). Previous to this terminal period, right hemiparesis had existed. The right arm throughout was most affected by spasms and paralysis, and exhibited increased reflexes. No aphasia or agraphia. The lesion, adhesive meningitis between dura and pia, with involvement of cortex of both central gyri on left side, as large as a quarter-dollar piece. There was another adhesion between membranes (without lesion of cortex) at the junction of the first frontal and precentral gyri. A third adhesion with lesions of the cortex existed over the gyrus hyppocampi. The pia was somewhat milky over the whole of the hemisphere. The left second and third frontal gyri, the right hemisphere and the cord were normal.

Holt[111] relates a case of pachymeningitis and leptomeningitis of several months' duration in a child 20 months old, characterized chiefly by persistent high temperatures, 102° to 105°, in spite of various forms of treatment. The spleen was large. No tuberculosis, although a twin of the patient and the mother had died of this disease. Warner and Beech[112] reports the case of a boy æt. 7 when first seen. Father syphilitic and mother neurotic. Soon after birth had sores on nates, and "snuffles." Was bright and well until a few months before examination, when he began to complain of headache, showed difficulty in various movements, was emotional and developed increasing dementia, for which he was confined, until his death at the age of 10 years, in an insane asylum. Lesions were false membranes under dura-mater over both hemispheres, extending well down toward the base, and adherent here and there to the pia. N. S. Davis, Jr.[113] has placed on record a case in which chronic meningitis about the posterior base of the brain produced peculiar symptoms. Male, æt. 60, complained of a cincture feeling about head, dizziness, drawing back of the head, thick speech, diplopia, drawling; contraction of cervical muscles constant. Later the legs became weak, more on right side. There was no pain. True bulbar paralysis was rejected by absence of muscular atrophy, and of falling forward of the head. There was no history of syphilis. The diagnosis of chronic meningitis about the medulla was made. Autopsy a few weeks later verified this statement. Adhesions between the dura and pia were found all around the medulla oblongata, bands of new tissue which had to be dissected away before the brain could be removed. The roots of the hypoglossus were most involved. Here and there upon the hypoglossus were opaque patches; on the left side there was adhesion between the cerebellum and the cerebrum. Moderate ventricular distension, but the basal spaces were largely distended with serum. Microscopic examination of the medulla revealed no lesions.

POLIOENCEPHALITIS.

In the course of an able and exhaustive lecture upon hemiplegia in children, based upon an analysis of 50 cases, Abercrombie[127] objects to Strümpell's theory of polioencephalitis as not supported by facts, the autopsies of old cases showing such secondarily altered cerebral scars that the nature of the original lesion cannot be made out. He knows of no case in which fresh polioencepalitis has been demonstrated. Smith,[115] in a paper upon cerebro-spinal fever, suggests that those cases of infantile hemiplegia which appear with high fever and convulsions may be cases of cortical encephalitis set up by the cerebro-spinal (microbic) virus. Against this view is the fact that most children survive the onset of epileptiform hemiplegia, while in its favor we have the result of Weichselbaum's[121] inoculation experiments on dogs. After inter-meningeal injection of cultures of diplococcus, localized encephalitis was among the lesions found. This, however, seems to have been more a common form of interstitial encephalitis with secondary breaking down of the nervous tissue than a true polioencephalitis. Kast[128] examined two cases of infantile hemiplegia at a relatively early period; one fourteen months and the other three years after the onset. In both cases the lesions were those of a diffused atrophy of the hemispheres. (Both affected in Case I.) Histologically there was interstitial as well as polioencephalitis, with marked vascular changes; though these were not sufficient to furnish an explanation of the sclerosis. The term polioencephalitis as employed by Strümpell is premature. (Kast gives quite a full summary of the lesions found by various recent observers in infantile hemiplegia.)

Wallenberg[150] also rejects Strümpell's polioencephalitis, as unjustified by post-mortem evidence. The frequent hemiplegias of infancy, often beginning suddenly with one-sided convulsions, and followed in time by contracture (spastic state), secondary epilepsy and athetoid movements, he considers as due to a variety of gross lesions, such as hæmorrhage, embolism, tumors (?). The treatment of the onset, or first stage, should be simply symptomatic. In the second or paralytic stage electricity is the principal agent, in the shape of faradization of the extremities. Galvanization of the head is contra-indicated,—though why, we cannot imagine. The epilepsy is to be combated by bromides, and for the post-hemiplegic disorders of movement, morphia, curare, physostigma may be tried. Nerve stretching is useless and dangerous.

INFANTILE HEMIPLEGIA.

Semeiology.—Abercrombie,[127] in his description of infantile hemiplegia, describes the paralysis as sometimes involving the face, and usually accompanied by marked contractures, and later by athetoid movements. Sensibility is very seldom affected. Sometimes there is deformity of the skull by atrophy of the half opposite to the paralysis. The leg shows a remarkable tendency to improvement. He does not mention the fact that aphasia is seldom permanent, even if the hemiplegia remains. Backwardness or idiocy is frequent. Infantile cerebral paralysis, strictly speaking, usually affects children under one year; onset by prolonged convulsions, often limited to the side which afterwards became paralyzed. It may require two or three separate seizures to produce hemiplegia, with or without aphasia. Strangely, the author does not include fever in the symptom-group. Secondary epilepsy is not sufficiently noted. Bernhardt[130] analyzed 18 cases. Besides ordinary symptoms, he notes that the right side was affected in more than two-thirds of the cases,—a fact which speaks strongly in favor of embolism as a pathological cause. Besides aphasia, and after it, many children showed defective speech (dysarthria), and many were also demented and subject to chronic epilepsy.

Pathology.—This is fully analyzed by Abercrombie, who contributes some autopsies of his own. He takes perhaps unnecessary pains to show that children are liable to hemiplegia from the same lesions as adults, viz., tumors, embolism. Tubercular meningitis shows itself sometimes first by hemiplegia. The acute infectious diseases (exanthemata) are occasionally followed by hemiplegia, and in this connection not a word is said of microbic infection and microscopic embolism. Cerebral and meningeal hæmorrhages are reckoned as varieties, although minute or punctiform hæmorrhages under the pia-mater are looked upon as more frequent lesions. The lesions in the typical cases of idiopathic cerebral hemiplegia are admitted to be little known; may be result of capillary hæmorrhage, or of thrombosis of veins or arteries. A true polio-encephalitis is very doubtful, and is not demonstrated by good post-mortem evidence. Embolism is the condition which the author considers most common, and best suited to explain the phenomenon.

Etiology.—Inherited syphilis is ranked first as a predisposing and active cause. Even arteritis obliterans may occur in young children, who are of course liable to gummata of the brain and its membranes. Violent coughing may lead to meningeal or parenchymatous hæmorrhage. Traumatism is mentioned, but we think it greatly underestimated by this author and others as a factor in infantile hemiplegia. Conditions of the blood are referred to, but only in the vague old-fashioned manner. At the meeting of the Clinical Society of London, West and Crocker[129] reported cases of hemiplegia and aphasia suddenly produced by violent paroxysms of whooping-cough.

Treatment.—Abercrombie[127] recommends warm or cold baths in the convulsive stage, according to the temperature of the patient, and says nothing of the use of chloral *per os* or *per anum* to arrest the convulsions (which in our opinion do much secondary mischief in many cases, and may prove fatal). Later the iodide of potassium should be given freely, with alkalies. The only local treatment is faradization of the non-contractured muscles. Bernhardt[130] is much more explicit. At first the treatment should be anticonvulsive and antiphlogistic; in the later stages much may be done by the administration of iodide and bromide of potassium; weak galvanic currents may be passed through the head, and faradism to the paralyzed muscles. Training and gymnastics, for speech and for the extremities, are of more importance, and in some cases orthopædic aid may be profitably invoked. None of these authors mention what we have repeatedly observed,—that cases of chronic epilepsy, secondary to the so-called sclerosis or lesion of hemiplegia, do not bear a thorough bromide treatment. Often their mental debility is greatly increased by doses which are not sufficient to control the convulsions. In such cases a solution of chloral and bromide will often do much more good, exerting a full anti-epileptic effect, with a minimum of cerebral depression.

HEMIPLEGIA WITH HYPERÆSTHESIA.

Drummond[131] relates a case of this rare combination. A lady, aet. 70, had sudden attack of numbness and paresis of the right side; the numbness was accompanied by intense prickling. On examination the whole right half of the body was found greatly hyperæsthetic and hyperalgesic, particularly to thermal impressions.

DISSEMINATED CEREBRO-SPINAL SCLEROSIS.

Oppenheim,[148] in an interesting paper, calls attention to some unusual features of this disease. He thinks that it is much more common in children than is generally supposed, having observed it at the ages of from 4 to 13 years; and he suggests that in many adult cases the symptoms might be traced back into childhood. He insists upon the facts that (1) the disease frequently affects the symptom-group of spastic paralysis; (2) that in such cases there are symptoms of disease of the optic nerves. In all his own cases, more than 50 per cent. presented optic symptoms, and in the five cases in which he had made an autopsy, all had sclerotic patches in the optic nerves, chiasm and tractus. Anæsthesia, while rarely permanent, is very often manifested as a temporary symptom. Temporary paralyses (facial, laryngeal, vesical and rectal) are also occasionally observed. The entire course of the disease is characterized by remarkable remissions and changes in symptoms. The author lays stress upon the fact (which we corroborate) that the syllabic speech is not present as often as the text-books would lead one to expect. The respiratory movements may be affected in a manner somewhat similar to that shown in speech. Involuntary laughter may be manifested. Finally he claims to have observed a new symptom of the disease, viz., rapid exhaustion of muscular power in non-paralyzed parts; a first movement being made with full power, a second with less, and so on to temporary inertia.

Moncorvo[138] has collected twenty-one cases of this affection occurring in children, aged from 16 months to 7 years. He considers that the most probable etiological factor is hereditary syphilis, endorsing in this respect the hypothesis of Marie and Jendrassik, that a perivascular and vascular lesion (syphilitic) is the starting-point of the various nodules of sclerosis. It is not claimed that all cases are due to syphilis.

Drummond[153] reports a case of a child, æt. 8 years, who presented a general flaccid paralysis with tremors and preserved reflex. Deafness, polyuria and glycosuria occurred later. Among the nodules of sclerosis there was found one quite large in the medulla, presenting on the floor of the fourth ventricle. Charcot,[238] in a clinical lecture, calls attention to various ocular symptoms observed in this disease. Common diplopia is rare, but frequently there is impairment in the associated action of the recti of the eyes, leading first to slight oscillatory movements and later to nystagmus. The phenomenon known as the Argyll-Robertson pupil is never met with, but the pupils are often very small, though active. Progressive amaurosis with color blindness is almost unknown, but there are attacks of temporary blindness without corresponding ophthalmoscopic changes, and occasionally the visual fields are concentrically reduced. The presence of spastic paraplegia should lead one to think of disseminated sclerosis. The same author, in a lecture upon the semeiology of the disease,[239] refers especially to the ataxic tremors observed in fully developed cases, and proposes a classification of tremors in general, based upon their rapidity or frequency per second, as shown by tracings. A first group is that of slow movements, from 4 to 5 per second, observed in disseminated sclerosis, paralysis agitans and senility. Second, rapid tremors, from 7 to 9 per second, which occurs in Basedow's disease, dementia paralytica, alcoholism, morphinism, and mercurial intoxication. Third, an intermediate group, with 5 to 7 vibrations per second, as occurring in the general trembling of hysteria.

DIFFUSED CEREBRAL SCLEROSIS.

Buss[135] relates in full a case of this lesion in a syphilitic child at 2½ years. The symptoms were microcephaly, the extremities were in the spastic or tetanoid state, sensibility apparently normal, syphilitic cutaneous lesions about nates, scrotum, etc. No trace of mental activity. Temperature often subnormal. Progressive emaciation. At death, weight only 9¾ pounds. Autopsy showed porencephaly, atrophy and induration of the convolutions; white substance of brain very scanty; some degenerative changes in the spinal cord (peri-ependymal). A striking microscopic lesion in the brain was the atrophy (or want of development?) of its myelinic fibres, both in the white and the gray substances. The degree of atrophy of this brain may be judged by the statement that while the brain of a child 2 years old weighs 1000 grams, that of this little patient, 2½ years old, weighed only 750 grams.

PRE-HEMIPLEGIC AND POST-HEMIPLEGIC TREMORS.

Stephan's[147] article on this subject is more critical than original, although it is based upon his thesis (Leyden, 1884). He maintains, after an analysis of all observations by other writers and himself, that involuntary choreic, ataxic and athetoid movements, as well as volitional tremors affecting a hemiplegic distribution, are due to a lesion in the thalamus opposite to the symptoms. This is in opposition to the theory (Köhler and Pick, Demange, Ricoux, Bidon) that these movements are caused by a lesion irritating the pyramidal tract at some point, but more in accord with the views of Charcot, Nothnagel and others, although these authors include the sensory division of the internal capsule together with the thalamus, as the seat of irritation. It should be added that Stephan's conclusion is in conflict with the more recent theory of the function of the thalamus, which would make it the organ of automatic expression or emotional movements. (Bechterew.)

HEMIPLEGIA WITH HYPERÆSTHESIA.

Drummond[131] records an instance of this rare symptom-grouping. A well-preserved woman, æt. 75 years, was suddenly seized with numbness and paresis of the right face, arm and leg, without loss of consciousness. In a few days the numbness developed into an intense prickly sensation with tenderness. The hand and foot were especially hyperæsthetic. A slight degree of contracture appeared; there was no aphasia, although speech was somewhat thick and nasal. The slightest touch on the tips of the fingers caused great pain, with an exaggeration of prickling, and the sensitiveness was greatest to thermic stimuli. The teeth on the right side were exceedingly sensitive. There was a degree of secondary contracture, and what may seem paradoxical, the deep and cutaneous reflexes were diminished on the right side.

PAINS IN THE LIMBS OPPOSITE TO A CEREBRAL TUMOR.

Brieger,[18] in the report of a case of pia mater sarcoma over the upper motor area of one side, notes pains in the opposite limbs. The same symptom was noted in Hun's[11] case IV; and Shaw,[133] of Brooklyn, presented a cerebral tumor to the New York Neurological Society in January, 1888, which had caused the same paræsthesia. In a case by Klippel,[110] of chronic meningo-encephalitis with Jacksonian symptoms, an aura of pain started in the right hand, and ascended to shoulder, when consciousness would be lost.

CEREBRAL SYPHILIS.

In the foregoing and in the following pages various cases of syphilis of the nervous system are recorded. The references now given are to more general essays. Braus,[137] in a monograph sums up his experience as follows: (1) The prognosis of cerebral syphilis is worse and more uncertain than that of any other organ. (2) Mercury acts very irregularly in different cases, and much of its success depends upon its early use. (3) The want of success of a mercurial treatment is no proof of the non-syphilitic nature of the lesion. (4) The truly syphilitic lesion of the brain gives rise to other changes, in time, of a secondary nature, which resist anti-syphilitic treatment, and sooner or later cause insanity. (5) After a time, or after repeated treatments, the mercurial influence no longer produces a favorable result. (6) The great majority of cases of cerebral syphilis come of the class of persons who are given to mental exertion. (7) The danger of cerebral syphilis lies in organic changes induced by the local syphilitic lesions. Braus' statements, we may assume, represent the special professional opinion of Aachen, where the author is a distinguished practitioner. That the method of treatment there employed is bigoted in its adherence to mercury and baths alone, few will deny. If the physicians of Aachen will employ the iodide of potassium in their cases, according to the American method (giving from 100 to 500 grains a day), the dismal prognosis given by Braus will be materially modified, and many patients will not be "sooner or later taken to insane asylums." Buttersack[145] reports the following case of syphilitic polydipsia and polyuria from Erb's clinic. A female, æt. 31, without history or symptoms of syphilis, suddenly developed excessive thirst, followed by polyuria, headache, dizziness and occasional neuralgia of the right trigeminus. No objective symptoms. Later there occurred occipito-cervical pain, tenderness of skull to percussion, deviation of tongue to right, right-sided ptosis with paresis of superior rectus and sphincter pupillæ; somnolence, vomiting. Very great amelioration followed two courses of treatment by iodide of potassium. No lesion of fundus oculi. Death from pneumonia. Lesion: leptomeningitis of base of brain and of spinal cord, neuritis of both motor oculi nerves and of spinal nerve roots. Syphilitic lesions of aorta and other vessels, and of the liver. The author and Erb consider the abnormal thirst as primary and the polyuria as secondary. Yet the reason for this view is not clear.

SYPHILITIC PSEUDO-PARALYSIS.

This condition of loss of voluntary muscular power through non-nervous lesions, occurring in infants, might aptly be called Parrot's disease (1872).

Lafitte[139] reports three cases cured by anti-syphilitic treatment. The lesions affect the epiphyseal ends of the bones, leading to separation of the cartilages. The periosteum and the adjacent muscles may also be involved. One result of this extremely painful condition is inertness or powerlessness of the affected extremities, which at first sight, without critical examination, may simulate a paralysis with hyperæsthesia.

The author's three cases were cured in 15, 30 and 55 days, under the use of Van Swieten's solution, Gibert's syrup aud sublimate baths.

PATHOLOGICAL INTRA-CRANIAL PRESSURE.

Falkenheim and Naunyn[140] have published the results of many experiments on this subject made since 1881. They treat of pathological pressure of the brain, or more correctly of the cerebro-spinal axis, produced by an increase in the pressure of the sub-arachnoid and intra-ventricular fluid. They recognize an idiopathic pressure produced (*a*) by increase in contents of and pressure in the vascular contents; (*b*) by anything which encroaches upon the cranial cavity (tumors, etc.). Anæmia or hydræmia is made out to be the most potent cause of the idiopathic or primary morbid pressure, although it is often due to lesions (inflammatory, etc.) which cause a retention of cerebro-spinal (ventricular) fluid. The symptoms of Marshall Hall's hydrocephaloid disease they consider to be due to increased pressure through hydræmia. When there is increased arterial tension, the pressure is usually easily equalized by increased absorption of the cerebro-spinal fluid; yet the authors do not consider the receptive capacity of the spinal cavity as very great in case of sudden increase in vascular contents or pressure. In the second category of vascular pressure, symptoms do not appear until the pressure of the sub-arachnoid fluid is made equal, or nearly equal, to the pressure in the carotids. The symptom-groups produced by sudden increase and by sudden fall of pressure in the cerebral vessels do not materially differ.

Broadbent,[143] in a lecture upon the relations of arterial pressure to cerebral symptoms, advances several interesting views, supported by relation of cases. Most of the argument is largely theoretical, but the following are points worth noting: Insomnia may be due to various causes, and among these are abnormally high and abnormally low arterial tension, as revealed by the wrist-pulse under the finger (or with the sphygmograph). In the first category, the arterioles are not strong enough to overcome the increased arterial pressure and a fine general hyperæmia results. This condition is best controlled by remedies which lower the arterial tension, and by the use of peripheral excitants such as hot baths, cold water compresses to the body, etc. In the second class of cases, change of air, tonics in general and remedies which increase arterial tension, will do good. In the author's experience, "in a very large proportion of cases of melancholia coming on late in life, the evidence of persistent high tension has been most marked."

Mercurial purgatives often do much good in these cases. The remarks upon the relation between the pulse and convulsions, are ingenious but hardly acceptable to the practical physician; several of the cases related appearing to be rather cases of uræmic eclampsia (in which high tension is a well-known symptom of the arterial, cardiac and renal lesions).

Albert,[146] in an elaborate critical review of the views advanced by Adamkiewicz and by Bergmann on intra-cranial pressure, sides with Bergmann in considering changes in the cerebro-spinal fluid (its amount and pressure) as an important factor.

Special importance is attached to clinical experiment upon a case of spinal meningocele in a child by Bergmann, as by pressure upon the sack various serious symptoms of cerebral compression—loss of consciousness, slow pulse (to 40), increased arterial tension and even Cheyne-Stokes respiration—could be produced. The article is largely argumentative and does not lend itself to brief analysis.

CEREBRAL AGENESIS.

Sachs[136] presented to the American Neurological Association the brain of a child two years old, in whom there had been no development of psychic activity, mental or kinetic. The child showed a number of reflex movements, and sensibility was well developed. The external configuration was normal. It was blind not long before death,—a blindness possibly due to retinal changes. No indications of syphilis or rachitis. Autopsy gave a brain which, while showing some œdema of the pia, was of full weight (1000 grams). Its gyral formation was rudimentary, the sulci very wide, and the island of Reil exposed (very much on the left side). The cortex was uniformly hard to the knife: its elements showed no trace of inflammatory or sclerotic change, but there was atrophy of the ganglion cells and of the peripheral and commissural nerve fibres. The appearances indicated want of development, and the author designates the case as one of "agenetic condition." A further report on the state of the great ganglia and mesencephalon is promised, and will be looked for with interest.

COINCIDENCE OF CEREBRAL TUMOR WITH BRIGHT'S DISEASE.

This case, reported by Drummond,[37] is just now particularly interesting, as observations are accumulating to show that during the course of Bright's disease, symptoms of local cerebral disease (even Jacksonian symptoms) may occur without corresponding gross local lesions in the brain. A man 42 years old had severe headache, prostration, loss of memory, mental dullness. His urine was scanty and highly albuminous. In a few days headache increased and delirium set in. There were no localizing cerebral symptoms and the optic nerves were normal. After increase in amount of urine, improvement occurred. A year later he presented himself again with very similar symptoms. No dropsy; urine albuminous, with casts. Died after increasing stupor. Autopsy revealed the large pale kidney of Bright's disease. At the anterior extremity of the right frontal lobe there was a gummy tumor as large as a fig, incorporated with the dura-mater and brain. From our knowledge of such tumors, it is doubtful if it was responsible for any of the symptoms, except perhaps the headache.

SUDDEN APPEARANCE OF SYMPTOMS FROM A CEREBRAL TUMOR.

Marquezy[132] presented to the Anatomical Society of Paris specimens removed from a man who retired one night dead drunk. The next morning he was found comatose, with right hemiplegia. At the autopsy the left temporal lobe was enlarged and its gyri flattened. It felt like a bag full of hazelnuts. On section there was seen a tumor with a yellowish caseous centre, surrounded by a fleshy congested part. It lay in the nucleus lenticularis and invaded the internal capsule in its knee and posterior part. Histologically the tumor was sarcomatous.

CEREBRAL CONCUSSION.

Albert[142] performed some experiments to elucidate the physiological pathology of this condition. Slightly curarized dogs were hammered on the head, according to the method of Koch and Filehne. Following the arrangement of Gärtner and Wagner, the veins receiving blood from the transverse sinuses were so prepared as to permit an estimate of the amount of blood flowing out, and at the same time the crural artery was connected with a kymograph. After the very first blow of the hammer there occurred an increase in the quantity of venous blood flowing out, coinciding with a rise of arterial pressure and retardation of the pulse; the outflow of blood continuing even after the arterial pressure had decreased. The author concludes that hammering of the skull (concussion) produces an irritation of the vagus, causing a dilatation of the central blood-vessels, very much in the same manner as obtained by electrical excitation of the cortex by Gärtner and Wagner.

PRIMARY ACTINOMYCOSIS OF THE BRAIN.

Böllinger,[134] of Munich, reports the case, which he considers unique (the only one out of 89 cases of actinomycosis in man). Female, æt. 26 years, complained of headache one year before death. A month later temporary paralysis of the left abducens. Eight months later, attacks of headache, diplopia, difficult speech, choked disks; occasional loss of consciousness. Temporary amelioration. Death after headache, vomiting and coma. Autopsy showed a tumor, size of a walnut, in the third ventricle, apparently developed from its choroid plexus. The surface was smooth, and its color pale yellowish-gray. There was internal hydrocephalus. Microscopic examination revealed the usual appearances of actinomycotic masses. The author thinks it probable that the germs were absorbed with raw goats' and cows' milk, which the patient was in the habit of drinking largely; from the intestinal tract it had reached the brain by embolism, through unknown channels. Only three cases of actinomycosis have been thus far observed in Bavaria.

OVERPRESSURE IN SCHOOL AS A CAUSE OF CEREBRAL DISEASE.

Madden,[141] of Dublin, calls attention to this potent factor in cerebral diseases of children. He insists upon the combination of overstudy, worry and malnutrition in the children of the poor, more especially, and suggests that besides reducing the amount of brain-work, the authorities would do well to furnish these children with nutritious food. While the author's remarks apply more particularly to the children of a miserably poor people, they are applicable to children in all countries. In this country particularly, we are convinced that many school children "break down" partly because of too much study, and study of too many subjects, but also because of scanty, hastily devoured breakfasts and nominal lunches, even in the better-off classes. The subject has many aspects which the author does not seem to consider, such as inherited cerebral capacity, conditions of the optic apparatus, etc., which complicate the problem and make it impossible for the teacher alone to discriminate between pupils and apportion work among them.

The late Dr. Edouard Seguin,[149] of New York, years ago insisted that this was a question which required the coöperation of the physician with the teacher.

EXCITABILITY OF THE CORTEX CEREBRI IN NEWLY-BORN ANIMALS.

Although this subject properly belongs to the domain of pure physiology, it is so intimately united with the physiology of cerebral symptoms in disease, that a summary of the most recent conclusions in this matter may prove interesting to the practitioner.

Bechterew,[144] of Kazan, has repeated the experiments of his predecessors, Soltmann (1876), Moüravoff, Rosenbach and others, with the following results:—

In newly-born puppies, the cortical centres remain inexcitable until the tenth day, when the different motor centres show excitability at slightly differing periods. A marked peculiarity is that at first these centres are easily exhausted by slight excitation.

Differences as to time were observed in different puppies, in some the cortex being inexcitable up to the fifteenth day. In the guinea pig the cortical centres are excitable at the time of birth. In puppies excitability of the cortex shows itself at a time when its ganglion cells are not fully developed, but when myeline forms upon the cylinder axes of the adjacent white substance. The increase of excitability in the weeks after birth goes on *pari passu* with the increase of myeline in the nerves.

These experiments tend to strengthen the theory (vide p. 6) that the cortical motor centres with their associated fasciculi, are really or directly motor, although in the normal completed mechanism their activity is probably usually developed as a sensori-motor or reflex action.

SPINAL DISEASES—VERTEBRAL CANCER.

A most valuable paper, from a diagnostic point of view, is by Amidon.[177] After referring to Cruveilhier's remarkable description of "paraplégie douloureuse," as caused by a hydatid cyst developed in the spinal canal between the dura and the laminæ of the vertebræ, he reports 3 cases, in 2 of which the diagnosis of vertebral cancer was verified post-mortem. He adds a summary of 21 other cases. 16 men and 8 women were affected; their average age was 45 years, 17 being the youngest and 79 the oldest. In only 5 had there been antecedent cancer; a family history of cancer in 2 cases. The morbid deposits (sarcoma and carcinoma) were found distributed as follows: in the cervical vertebræ 6 times; in the dorsal 17 times; in the lumbar 22 times; in the sacrum 3 times; and 3 times in all the vertebræ examined. The third lumbar vertebra was most often diseased (9 times). In many cases there were deposits in other organs. The earliest and chief symptom of this affection is pain, acute or dull, in the spine, and along various nerves in relation with the seat of vertebral disease; it may be very acute and accompanied with local spasm; it is often bilateral, though in some cases unilateral and very localized. The character is that of a referred pain, often worse at night. Deformity of the spine is sometimes present, but never as distinctly angular as in Pott's disease. Paraplegia also occurred ten times. The chief difficulty in differential diagnosis is between the disease in question and Pott's disease, or acute caries of the spine. The author presents the following table of symptoms to aid in this diagnosis:—

	CARIES.	MALIGNANT DISEASE.
Age	A disease of youth.	A disease of adults.
Antecedents	Tubercular.	Malignant.
Local tenderness and pain.	Rare.	Common.
Neural pains	Later.	Earlier.
Deformity	More frequent and angular.	Frequent, and seldom angular.
Anæmia	A late symptom.	An early symptom.
Emaciation	A late symptom.	An early symptom.
Cachexia	If present, a very late symptom.	An early and constant symptom.
Œdema	Rare; if present, anæmic or renal.	Frequent, and from obstruction.
Enlarged glands.	Rare, and, if present, general.	Common and localized.
Abscesses	Present.	Absent.
Temperature	Hectic.	Seldom elevated.
Location of disease	In a large proportion dorsal.	In a large proportion lumbar.
Other organs involved	If any, general tuberculosis, predilection for nerv. system or lungs.	All organs, most often the liver.

True paraplegia, when it occurs, of course due to compression myelitis, and the same in both.

The author does not mention spinal rigidity, which we consider a very valuable symptom as against intra-spinal tumors; this symptom being common to Pott's disease and vertebral cancer. Another omission is that in some cases the lesion will be overlooked at the autopsy unless the vertebral bodies are split open. Case 12 came very near being misjudged on mere inspection of the vertebræ.

Putnam[178] reports a case of cancerous disease involving the intra-abdominal nerves and producing the symptom-group of paraplegia dolorosa.

PERI-PACHYMENINGITIS.

Morse[180] reports a case of this rare affection. Male, of neurotic family. When 19 years of age was exposed to a cold rain for several hours, and some nine hours after complained of pain in right side of the neck, diagnosticated as abscess, severe pain, fever, and about four weeks later, a sudden paralysis on the right side of the body. In the course of a few months this and all other symptoms were relieved, the swelling in the neck becoming harder and smaller before disappearing, patient remained well for fully three years, when he was attacked with headache, vertigo, insomnia and weakness of the back. On fifth day somnolency noted, and after it prickling sensation in the dorsal region. On examination 11 small swellings (abscesses) were found in the region of the right rhomboid muscles: one at level of second dorsal vertebra was opened and discharged offensive pus. From another abscess on the next day a small piece of bone was discharged, followed by repeated convulsions, coma and death on the seventh day. Autopsy: abscess found in location indicated, with one long sinus extending downward 5½ inches. "Surrounding the dura-mater there was a mass about 3.7 mm. in thickness, extending from the point of the opening of the sinus to the fourth dorsal vertebra." The dura, pia and cord seemed healthy, except that the last appeared to have been somewhat compressed (no paralysis). Strangely enough nothing is said of the condition of the vertebræ, and the head and cervical spine were not examined. (A note from the author informs us that "the vertebræ were carious to some extent.")

HYPERTROPHIC CERVICAL PACHYMENINGITIS.

An abstract of a lecture by Charcot[181] contains an account of a case completely cured. A female (age not stated, but from context must have been over 40 years old) first noticed rheumatoid pains in the neck, shoulders and arms. A few months later the arms became paralyzed, with some atrophy. Some seven or eight months later the legs became weak. When admitted to the Salpétrière she presented a spastic paraplegia, with flexion of the legs and exaggerated reflexes. Under the use of the actual cautery over the cervical region, electrization and the internal use of iodide of potassium, marked and progressive improvement appeared, but the lower limbs were still contractured, and examination by a surgeon (Terrillon) revealed the existence of strong fibrous bands under the joints. These were cut and the limbs placed in a fixed apparatus. Two months later the patient was well. Charcot defines in this lecture the three stages of the disease, viz., (1) the neuralgic, a painful stage (pains in neck and upper extremities); (2) cervical paraplegia (arms) with atrophy of a variable number of muscles and occasionally contracture; (3) spastic paraplegia with flexed limbs and often formation of fibrous hands about the joints. Remak[236] related to the Society of Internal Medicine of Berlin, an incomplete case of this sort (neuralgic stage absent) characterized mainly by an atrophic paralysis of muscles innervated by the median and ulnar nerves on both sides (main-en-griffe), some impairment of sensibility and DeR. in the atrophied muscles. There was tenderness over the second and third cervical vertebræ, and slight rigidity. Under iodide of potassium and galvanization (6 ma.), a complete cure was obtained in two months.

SPINAL HEMIPLEGIA.

M. Rosenthal[175] reports three cases: (1) A girl, æt. 18 years, who had rigidity of the neck and swelling about the region of the third, fourth and fifth cervical vertebræ. Passive movements of the head were painful. The illness began with fever and pain in the neck. Four months later paralysis of the body appeared and progressed. When seen by Rosenthal in September, 1883, besides the above local cervical symptoms, there was atrophic paralysis with increased temperature and perspiration of the right arm and leg; the arm was fully paralyzed. DeR. was present only in the muscles of the thenar eminence, although all the muscles of the extremity were much wasted. On the left side, from the shoulder down, to the median line, there was complete anæsthesia. Recovery (under galvanism and K I) took place in 14 months; motor power returning centripetally, sensibility centrifugally. The order of recovery of forms of sensibility was as follows: contact, tickling, pain, and last, temperature. Heat was appreciated sooner than cold. Rosenthal considers the lesions to have been a one-sided (right) pachymeningitis externa, in connection with spondylitis. (2) Woman, æt. 36, after a fall on the back complained of pains and stiffness, later of paralysis in the left extremities. On examination three months after injury (1881), there was deviation, rigidity and sensitiveness about the third to sixth dorsal vertebræ (kyphosis and intercostal pain not mentioned). The left arm and leg were paralyzed, the latter more; sensibility was present and patellar reflex increased. On the right side there was distinct though incomplete anæsthesia, while voluntary movements were normal; patellar reflex subnormal. Under cauterization, K I and galvanization, a complete cure was obtained in 9 months. Rosenthal's diagnosis of spondylitis will not be easily accepted. The occurrence of symptoms in the arms cannot be explained by dorsal spondylitis and pachymeningitis. (3) Male, æt. 32 years; pain in cervical spine and consequent stiffness; five months later paresis of right leg and arm observed. Examined in sixth month of disease (1884); patient is emaciated, is unable to move head without assistance of hands, cervical vertebræ tender and almost fixed; a soft swelling (abscess) presents on right side of neck. The right arm and leg are almost completely paralyzed; some movement at the hip, and the shoulder moves a little from action of serratus; sensibility normal, patellar reflex increased. On the left side of the body motility is normal, tactile sensibility is reduced, while the sensibility to temperature and pain is entirely abolished. Left patellar reflex subnormal, or at least less than on right side. Later complete cervical paraplegia developed, with bed-sores, etc. No autopsy was obtained. Rosenthal adds a case of hysterical paralysis in a woman of 25, having the same distribution. All these cases are marred by astonishing omissions of observation; the pupils and facial circulation are not studied, no peripheral temperatures are recorded, the secretions on the two sides of the body are barely mentioned (once), the condition of the muscular sense (one of the most important symptoms) is noted only in the hysterical case. Delmas,[241] a French army surgeon, relates the following case: A healthy man, æt. 25 years, received a stab wound on the left side of the neck. The blade, narrow and very sharp, entered at the anterior edge of the trapezius and penetrated inward as far as the spinal cord (?). There occurred immediately paralysis of the left arm and leg. As in Sach's case, there were no symptoms on the opposite side. The temperature was slightly higher (in axilla ?) on the left side than on the right, but not constantly. No pupillary or facial symptoms noted. On the thirtieth day a difference in circumference of arms of about 2 cent. (4–5 in.) was observed in favor of the healthy side. In two months the patient was well. This rapid recovery, the absence of crossed symptoms and of facio-ocular symptoms, would suggest either that the left lateral column of the cord was simply bruised, or that the case was one of traumatic hysterical paralysis. Sachs[212] reports a rather anomalous case of a tubercular tumor involving the left half of the cord at the level of the sixth and seventh cervical segments, starting near the posterior roots. The symptoms for eight weeks were unilateral, pain in left arm and hand (in ulnar distribution mostly), with hyperæsthesia and paresis. The left leg showed slight paresis, hyperæsthesia, increased knee-jerk and ankle-clonus; increased paresis of left leg. At no time in this first period was there anæsthesia or indeed any symptom on the right side. The muscular sense was very defective on the left side, and preserved on the right. In the eighth week symptoms of transverse myelitis set in, with total paralysis and anæsthesia below the neck.

TUMORS OF THE SPINAL CORD.

W. Pasteur[171] presented to the London Pathological Society two cases of diffused sarcoma of the pia-mater. (1) Woman, æt. 22 years, symptoms of a cervical pressure myelitis, choked disk, paralysis of both external recti, occipital and spinal pain, stupidity. Lesion: a soft gray mass of new tissue surrounded the cord, mostly on its posterior and lateral aspects. It extended from the nerves of the cauda equina; and upward it had spread over the pons and medulla, completely investing both sixth nerves. It was a subarachnoid, round-cell growth, and here and there it extended within the cord along the trabeculæ. The nervous tissue was normal.

(2) Child (female) æt. 4½ years; fall down steps two months before; followed by strabismus, and paresis of legs and arms, and, two weeks before admission, sudden blindness. There was pain in the back. The spinal cord was surrounded by a thickened layer of new growth, especially developed on the posterior aspect. It extended upward to the under surface of the cerebellum, whose middle lobe was almost entirely replaced by new growth. It is not stated how far down the growth reached. Its structure was like that of first case. In neither case was there anæsthesia. Pains and paresis gave expression to the pressure and irritation.

Taube[202] reports a case of a very rare form of spinal tumor, viz., lymphangioma:—

A female, 46 years of age, complained two weeks before admission to hospital of pain and weakness in the right leg, and later in the left; numbness began in the toes and extended gradually upward; the legs became paretic without spasm. Later there was common paraplegia, with anæsthesia up to the level of the sixth intercostal space, and with increased reflexes. The electrical reaction was normal in thighs and approached to DeR. type in the flaccid legs. Sensibility of abdomen varied at times. A painful girdle feeling existed at level of sixth ribs. There was tenderness over the seventh and eighth dorsal spines. Death in fourteen weeks.

Autopsy showed an ovoid tumor overlying the posterior aspect of the cord, between the sixth and seventh dorsal nerves. It was about the size of an English walnut, and lay under the arachnoid. The spinal cord was flattened. Histologically the tumor consisted of numerous cystic cavities, showing here and there distinct endothelial layers; many of the cysts contained coagulated lymph. The heavy connective tissue stroma was partly sarcomatous and rich in ectatic blood-vessels. A small tumor of same sort was found in the pia above the main growth. The spinal cord was disorganized at the point of compression; above there was degeneration of the columns of Goll, below of the pyramidal tracts to the end of the cord.

PRESSURE MYELITIS. (TUMORS, ETC.)

Grasset and Estor[170] relate at length a case of myelitis of the upper cervical enlargement caused by unrecognized Pott's disease. A non-syphilitic, temperate man, æt. 37 years, two months after a severe wrench of the right wrist, experienced neuralgic pain about the right shoulder region, without arthritis. Later paresis of right arm muscles appeared, with dilated pupil on right side, and a zone of hyperæsthesia in the lower level of distribution of the cervical plexus. After a remission, these symptoms returned, with similar ones on the left side. Both arms were paretic (more than the hands), the neck behind was tender and motion limited; pressure on cervical spinous processes caused pain in right shoulder. The deltoid was the only muscle showing marked atrophy and partial DeR. Reaction normal in all other neck muscles. Five months later an acute febrile attack with enlargement of thyroid gland occurred; some pulmonary symptoms. Later partial paralysis of legs, without anæsthesia; patellar reflexes increased. Eleven months after onset the hyperæsthestic zone became normal, but below the third intercostal space there was complete anæsthesia and paralysis (arms also). These symptoms appeared rather suddenly, and were followed by death in 24 hours. Autopsy showed a reddish softening of the cervical enlargement in a length of about two inches. The bodies and articular facets of the third and fourth cervical vertebræ were diseased, and a tumor-like formation presented (one swelling on either side of the median line) upon the fourth vertebra, on the posterior aspect of its body under the posterior common ligament, compressing the anterior aspect of the cord. There was no pachymeningitis. The lesion of tubercular thyroiditis, with degeneration of various lymphatic glands connecting (?) with the vertebral disease were found. No bacilli were discovered by Ehrlich's staining. It is singular that a nearer approach to a correct diagnosis was not made. The senior author was inclined to look upon the case as one of cervical myelitis from the injury to the wrist (!). He states that "all the symptoms of a cervical myelitis" were present, which can hardly be accepted, as the neuralgic and paretic symptoms were limited and one-sided to begin with, and the spine was tender. The neuralgic pains, the stiffness of the neck, the irregularity of paralysis, and the dilated right pupil should have led at least to a diagnosis of a compression lesion of the cord, either osseous or dural. It would have been impossible, perhaps, to have decided whether the lesion was a true tumor or Pott's disease. The test of pressing forcibly on the vertex does not seem to have been tried. (Compare vertebral cancer.)

Ryan[175] reports a case of paraplegia from Pott's disease rapidly improving under large doses of iodide of potassium,—from 3 to 55 grains three times a day. The patient was a child æt. 6 years; from a condition of extreme paraplegia she improved so as to be able to take a few steps without support (in five months). The discussion which followed (in the Cincinnati Academy of Medicine) shows how little the profession generally know of the tolerance of the iodide of potassium by children. The case is rendered of doubtful value by the facts that an apparatus was also used (even previous to the giving of the iodide), and that the doses of the remedy were too small, except toward the last, to be of much use.

Taube[202] gives a case of pressure myelitis due to lymphangioma of the pia-mater.

Harris[210] relates at length an interesting case of sarcoma of the dura-mater (or peri-pachymeningitis ?) which involved and compressed the cord chiefly at level of sixth to eighth dorsal vertebræ, but also extended downward outside of the dura, to the lumbar region, almost encircling the cord. The symptoms were the usual ones of pain in the side and round about body at level corresponding to growth (and treated as a gastric pain !) followed by paraplegia and very early bed-sores (in 48 hours). The interest of the case lies in the fact that paraplegia developed and became complete in less than 24 hours.

SYRINGO-MYELIA.

A monograph by Wichmann[203] on this subject (or gliomatous cavity formation in the cord) appeared in 1887. He relates the following case. Male æt. 34 years at time of first symptoms in 1877. These were pains in neck, breast and arms, lasting a few weeks. Two or three similar but longer attacks occurred at intervals, and in the winter of 1879–80 the legs were very paretic (support needed), and arms weak. Improvement again occurred, so that he was able to walk in the summer of 1880. A fourth relapse then occurred, followed by partial improvement. In 1882 sharp pains in neck, head and teeth, and in lumbar region extending into the legs. Later spasms in lower back and legs, pollutions and incontinence of urine. When seen at Wildbad by Dr. Renz in 1885, patient presented a spastic paraplegia, with great increase of reflexes (foot clonus present) and reduced sensibility; no atrophy. The upper extremities were paretic and somewhat atrophied (the right more). Abdominal muscles and bladder paralyzed. Death by apnœa 8 years after onset of this remarkably remittent disease. Autopsy showed a gliomatous tumor 4 cm. (1 3-5 in.) long in the middle of the cervical enlargement; the gliomatous tissue extended downward, in the shape of a central cavity, to the lumbar enlargement. The author presents an analysis of 33 cases he has collected. Of these 22 were males, 11 females; average age at onset 33–34 years; average duration of disease 3 to 4 years (extremes 2 and 8 years). The entire cord was involved in 6 cases; partially and more especially the enlargements, in 27 cases. As regards the extension of the lesion transversely (as seen in cross sections), the postior columns were involved 18 times; the posterior horns 8 times, and the anterior 5 times. In 17 cases the cord presented a tumor like swelling or enlargement, most common in the cervical enlargement; in many cases the gliomatous growth extended beyond the limit (vertically) of the swelling. Symptoms: These were absent in several cases. Almost always there were pains in back and limbs, usually worse in the latter. Partial anæsthesia was observed in 17 cases; total and general in two cases (in one with fragilitas ossium). Paralysis is the most frequent symptom, irregularly distributed, mostly in the legs; the right leg and left arm were most often affected. In 11 cases there were symptoms of lesion of the medulla oblongata, and in a few cases lesion of cranial nerves. One of the most striking features of this disease, almost a characteristic, is the remittent type of the symptoms, with occasional remarkable improvement. Baumler (Anna)[229] has published an exhaustive essay on this disease, including one case which she observed in Eichhorst's service at Zürich, and examined microscopically. The analytic study of 112 cases is presented.

MYELITIS.

Recoveries from "myelitis" and "meningo-myelitis" are reported by Steven;[166] by Fairbank[169] (this is so badly reported, without electrical examination, that diagnosis is next to impossible: it was most likely multiple neuritis); by Lewtas,[204] a case of syphilitic meningo-myelitis (reported as acute ascending paralysis).

Glynn[224] relates a case of paraplegia from Pott's disease ("regarded as one of transverse myelitis") in which all the symptoms, including large bed-sores, of complete paraplegia were present for ten months. The curvature was in the cervico-dorsal region. In the eleventh month improvement began (without active treatment) and progressed to a stage of partial recovery with spastic legs, and later to complete recovery. The lesion was more likely pachymeningitis causing pressure, as such perfect recovery from a transverse myelitis, in the strict sense of the term, is hardly admissible. McCall Anderson[237] reports two cases in which the lumbo-dorsal and the upper dorsal region of the cord were affected respectively. The first case recovered in 3 months after treatment by ergot *per os*, and atropia subcutaneously. The second case had, in addition, flying blisters to the spine, was fed through the nose during a period of dysphagia, and was well in two months.

Pathological Anatomy.—Dr. Railton showed to the Manchester Pathological Society sections from the spinal cord of a man who had died after complete (sensory and motor) paraplegia with retention of urine. The constriction band was at a level two inches below the umbilicus. A part of the lower dorsal cord was completely disorganized, in a transverse way. Above the main lesion there was found extension of the myelitis in the gray matter for a certain distance (not given), secondary changes in the columns of Goll, of Gowers and in the ascending cerebellar tracts. Below the focus there was degeneration of the pyramidal fasciculi and some slight changes in the gray matter. The blood-vessels (in the cord?) are said to have had thickened walls. The patient had had syphilis. Nauwerck[217] made a critical histological study of a case of so-called transverse myelitis involving the cord from the fifth to the eighth dorsal nerves. The lesion was in reality a softening. The degeneration of nerve fibres was accompanied by slight increase in the neuroglia (sclerosis). At one point (seventh dorsal nerve) the neuroglia also was disintegrated. In no part was there evidence of inflammation. The small arterioles near the focus showed thickening and hyaline changes of their coats; some vessels were blocked by pale thrombi; others showed endarteritis. It is claimed that this is the first case in which arterio-sclerosis has been shown to be the cause of spinal softening, and its occurrence in a subject only 24 years old is attributed to a preceding typhoid fever (?). The usual ascending and descending degeneration was found.

SPINAL LESIONS PRODUCED BY LIGATURE OF ABDOMINAL AORTA.

Experiments upon this question are of highest interest for human pathology, more especially in relation to so-called "myelitis" and "poliomyelitis." The gross symptoms (paraplegia) following ligature of the aorta were first observed by Steno (1667), Lecat (1765), Bichat (1812) and by many others since. In 1884 Ehrlich and Brieger made observation upon the histological alterations of the spinal cord after this operation; and recently Singer[188] has repeated the experiments. In animals (rabbits?) killed 24 or 36 hours after ligature of the abdominal aorta below the renal arteries for one hour, he found granular degeneration of the multipolar cells. On the fourth day there were destruction of myeline and of cylinder axes in the anterior horns, the antero-lateral columns and the anterior nerve roots. After five weeks, section showed shrinkage of the entire gray substance, absence of multipolar cells; the antero-lateral columns and the anterior nerve roots (with the exception of a few fibres), were degenerated. The peripheral nerves were extensively degenerated and the muscles sclerosed. On the other hand, the posterior ganglia, the posterior roots and the posterior columns were normal, although during life anæsthesia had been present. In the anterior commissure were a few normal fibres. It follows that the arterial supply of the posterior spinal system is not directly dependent upon the abdominal aorta (lumbar branches). [It is possible that further experiments and researches in this direction will help to clear up the pathogenesis of local "myelitis," and of polyomyelitis.]

POLIOMYELITIS.

Semeiology and Diagnosis.—Cordier[221] read before the Société des Sciences Médicales of Lyon a report of an epidemic of infantile spinal paralysis. [Paper not published: abstract will be given in ANNUAL of 1889.] Oppenheim[222] reports a case of chronic anterior poliomyelitis. Female, æt. 52 years. gradually developed a flaccid paralysis of all the extremities and of many truncal muscles. The atrophy affected the muscles *en masse*, and not in a fascicular way. No sensory symptoms. In one leg the patellar reflex was present, while the quadriceps still reacted to the will, and no electrical changes were present; it disappeared as these signs and paresis became evident. In many muscles various degrees of DeR. were present, but later all electrical excitability ceased. Bladder unaffected. No bulbar symptoms. Disease lasted three years.

Pathological Anatomy.—Sharkey[155] examined the nervous system in a man, æt. 60 years, who had had infantile spinal paralysis of both legs from infancy. The brain did not show any evident atrophy. The lumbar cord was much wasted, all of its white columns were much shrunken, but the antero-lateral had suffered most. The anterior horns presented a fine fibrillary structure, in which few ganglion cells remained. There were innumerable corpora amylacea, and the blood-vessels presented a hyaline appearance within dilated perivascular canals. The paralyzed and atrophied muscles consisted almost wholly of fat; here and there were seen some striated fasciculi. No mention is made of ascending degenerative changes. Potherat[184] describes at length the naked-eye appearances of the atrophied muscles and bones of the upper extremity in a case of poliomyelitis occurring at the age of 18 months, and removed after the patient's death from phthisis at the age 46 years. The details are interesting, but as a microscopical study of the specimens and of the cord is promised, a further analysis is unnecessary. It is well to mention that the brachial plexus and its branches on the two sides appeared of the same size. Parmentier[185] publishes a similarly incomplete observation. Atrophic paralysis of the right upper extremity, supposed to have been caused by a fall on the shoulder at the age of 15 months. Autopsy at 43 years. The motor regions of the brain showed no difference on the two sides. The anterior roots of spinal nerves on the right side in the cervical region were smaller than on the left, although, as in the preceding case, the brachial plexus and large nerves appeared equal on both sides. In Oppenheim's case[222] of chronic poliomyelitis, there were found degenerative changes in the various muscles, in the motor nerve fibres and the anterior roots. In the anterior horns the ganglion cells were nearly all absent, and there were abundant spider cells.

Genesis of Deformities (*Contractures*).—Under this title Lorenz[182] re-examines the various theories advanced to explain the contractures often observed after poliomyelitis, and concludes that the explanation of Volkmann is sufficient. This is that the main cause of deformity is the unbalanced weight of the part, which has full play owing to paralysis of some muscles supporting it. This is best shown in the foot, which, independently of muscular support, is badly balanced. When the anterior tibial muscles are paralyzed, the weight of the foot tends to produce a pes equinus, equino-varus or equino-valgus. When the posterior tibial muscles are paralyzed, on the other hand, no contracture occurs. Werner's suggestion that constant slight contraction (tonus) occurs in the non-paralyzed muscles and assists in producing deformity, is also accepted by Lorenz; who adds that in later stages a degree of shortening occurs in totally degenerated muscles, which would explain the rare cases in which there is contraction with paralysis of all the muscles of a part.

HÆMATOMYELIA.

Leyden[228] reports at length a case of this rare affection, and treats of the subject in general. A woman, æt. 28, immediately after an easy confinement, had continued backache and complete paralysis of the right leg. Soon sensibility was lost. On the second day the left leg became weak and on the third complete paraplegia was established, with loss of reflexes and electrical excitability. Death in about one month. The diagnosis was acute or apoplectic myelitis. The autopsy revealed a hæmorrhage in the cord extending up and down in the lower dorsal and upper lumbar segments, in the deeper part of the posterior columns. There were surrounding softening and disintegration, total or transverse, for a space of two inches just above the lumbar enlargement. There was also the usual ascending and descending degeneration. The all-important question whether in such cases the hæmorrhage is primary, or constitutes a complication of a preceding myelitic or necrotic process, the author decides in favor of the first hypothesis. For him there is such a disease as primary spinal hæmorrhage or hæmatomyelia.

ACUTE ASCENDING PARALYSIS (LANDRY'S).

Semeiology and Diagnosis.—Douie[156] relates the following case without autopsy: Woman, æt. 50 years, in good health, complained of tingling and weakness in left hand one morning. By evening of same day both arms paralyzed, though hands could be moved a little. Next day the paralysis of upper extremities was complete and the legs were weak. On the fourth day all extremities were motionless. On sixth day whispering voice and dyspnœa; death on seventh day. No fever occurred; there were no sensory or trophic changes: no loss of electrical practice; the reflexes were abolished. No paralysis was observed above the shoulders. Möbius[157] reports a case in which motor paralysis of an ascending form occurred after whooping cough. Child, æt. 3 years, had whooping cough for six weeks. As this declined the child's legs became powerless and flaccid, with absence of knee-jerk but with preserved cremasteric and abdominal reflexes. No changes in electrical reaction of nerves and muscles. Three weeks later the legs were better, but the arms were almost powerless; the cervical muscles were very weak, but the intercostal and abdominal muscles remained normal. The diaphragm lost power and some bronchial catarrh with cyanosis set in. At about the twenty-fourth day from first examination decided improvement appeared, and convalescence progressed to complete recovery by the forty-sixth day. At no time were the bladder and rectum affected, and there was no anæsthesia. The author considers the lesion to have been a multiple neuritis limited to the motor nerves, an opinion which we consider untenable, in view of the facts that sensibility and electrical reaction were normal. Only a neuritis of the anterior spinal roots would be admissible, and even on this hypothesis how explain the escape of the twelve or fourteen anterior roots of the dorso-lumbar segment? Pitres and Vaillard[158] have fully described a case in which the lethal progress was extraordinarily rapid. A healthy man, æt. 22, was ill with slight bronchitis, fever, diarrhœa and prostration (simulating a short attack of typhoid fever). After three days of absence of fever, it appeared again and was found to be due to double slowly ascending pneumonia. At 4 A.M. on the twenty-fourth day of his illness, patient awoke with complete loss of power and sensation in both legs; the inspiration was somewhat difficult. In four or five hours the abdominal and intercostal muscles became paralyzed, with absolute anæsthesia to nipple level; soon the arms became affected, the voice weak and expectoration impossible. At 3 P.M. the body below the neck was completely paralyzed and anæsthetic; the diaphragm and cervical muscles acting. Faradic contractility was then found reduced. Death late in the evening. The paralytic attack thus appears to have run its course in from 12 to 16 hours. Négrié[213] reports a case occurring after mild uncomplicated rubeola. Child, æt. 3 years, of excellent constitution; at tenth day, in convalescence, weakness of legs and arms was observed; cervical muscles weak, headache, pains in the extremities; axillary temperature 39.5°. Next day general paralysis below head; patellar reflex abolished, plantar reflex nearly so; perspiration normal; pupils normal; sensibility unimpaired; bladder and rectum normal. On fifth day death by apnœa. No autopsy. Mann[215] gives a very full report (marred by failure to make histological examination of peripheral nerves), of a case presenting following symptoms:—

Healthy male, æt. 48 years, free from neurotic antecedents and syphilis. Onset by sense of cold in legs, tingling in feet, cramp-pains in legs. On third day legs weak; increasing paralysis; no further sensory symptoms. When examined on eighth day, presented absolute paralysis of legs and trunk, partial of arms; legs œdematous. Sensibility perfect. Muscles were flaccid but reacted normally, as did the nerves, to faradic current. No reflexes could be obtained. Bladder acted, and the urine was albuminous. No fever. Respiration embarrassed; pupils normal. No tenderness of nerves or muscles. On the ninth day the arms were completely paralyzed, the breathing more and more difficult, and death occurred with delirium or convulsion, from apnœa. For autopsy *vide infra.*

Pathology and Etiology.—Pitres and Vaillard[158] found the spinal cord normal to naked eye and microscope. The spinal nerve roots showed few alterations. All the peripheral nerves, on the other hand, showed widespread lesions. The myeline was more or less dissolved, the cylinder axis was absent in many fibres, of which nothing remained save an empty sheath of Schwann. The protoplasmic nuclei of the fibres were unchanged. It would appear that the nuclei had undergone a sudden liquefaction, and that there were none of the active changes observed in the Wallerian degeneration. The authors, in a review of the whole subject, incline to the view that ascending acute paralysis is always a multiple neuritis. Their own case evidently was due to infection from the acute diseases which affected the patient, viz., broncho-pneumonia and typhoid (?) fever. In Mann's case[215] autopsy showed perfectly normal nerve centres. A careful microscopical study of the cord, medulla, crural and motor region of the brain, made by Robinson, of Owens College, showed absolutely no lesion of these parts, in their various elements. The lymphatic glands not enlarged; liver and kidneys congested. Peripheral nerves not examined. The author inclines to the theory (held by Landry, Westphal and others), that a toxic agent, possibly a ptomaine, is the active cause of the paralysis. It may act as an "elective" poison, inhibiting the motor function of the cord. In his case, Mann rejects multiple neuritis, because of the absence (at the height of the disease) of numbness, pain and tenderness, and because the faradic reactions of nerves and muscles were normal on the eighth day: a good argument.

POSTERIOR SPINAL SCLEROSIS.

Semeiology and Diagnosis.—Kayer[191] has analyzed the symptoms presented by 117 cases in Mendel's clinic, and the following are some interesting results. Impairment of vision occurred in 35 per cent.; Argyll-Robertson pupil in 66 per cent.; patellar reflex preserved in 8 cases (nearly 6 per cent.). DeWatteville reports[198] as a novelty, paresis of convergence in a tabetic patient, æt. 36. There was no diplopia and as the pupils were equal, it seems more than likely that this was an ancient weakness of the interni (left internus most). The case was still in the first stage, with only paresis, Argyll-Robertson pupil and absence of knee-jerk as symptoms. Porter[218] presented sections of the spinal cord of a patient who had died of a cardio-pulmonary complication after having had symptoms of tabes only eight weeks. These symptoms were sharp pains in legs and arms, sensation as of walking on sand. Examination showed absence of patellar reflex, and ataxic gait. The cerebral and spinal meninges were thickened and the posterior columns of the cord showed sclerosis in both their fasciculi. As recorded, this case will not stand as one of acute posterior spinal sclerosis.

The history is extremely deficient, no mention is made in it of syphilis, and the pains are not well described. In the examination no statement occurs as to condition of eyes or sensibility, or of coördination of hands. To any ex-interne of a large hospital, it will appear probable that the attention of the staff was absorbed by the cardiac and pulmonary symptoms, and that the history was not taken with care. Besides, we have known most intelligent victims of tabes, who, after several weeks of intercourse with the physician, would finally trace back their first pains as "only rheumatism" several months and years prior to the date given in the first interview. Hutchinson[225] relates in a somewhat loose and chatty manner the history of some nine cases of "ataxy" or of impending ataxy. Of these, four are quite certainly not cases of posterior spinal sclerosis, but rather of syphilitic lesions of the cerebral nervous system, or of simpler degenerative changes. Charcot[238] states that in exceptional cases tabes may reach its third stage in 15 days. (The reviewer has published a case in which ataxia was complete before the twentieth day, but it was a case of pseudo-tabes, probably from double sciatic neuritis.)

Laryngeal Paralysis with Crises.—Wegener[168] describes two cases of this complication, and gives a summary of other cases. In all (?) the laryngoscopic lesion was the same, viz., paralysis of the abductor of the vocal cords. Cardiac symptoms of various sorts occur in the course of the disease. Leyden[192] refers to the coincidence of valvular lesions; to the occurrence of crises-like attacks of oppression of breathing, feeling of anxiety, pain in the precordial region and in the left arm—in brief, anginoid symptoms. A rapid pulse is also frequently met with. These symptoms are referable to lesions in the nucleus or in the trunk of the pneumogastric nerves. The paroxysm may or may not coincide with gastric crises. Vulpian[193] met with anginoid attacks in a case in which autopsy showed no cardiac lesions. Crises in general have been studied by Eckert[200] under the direction of Leyden and Westphal. The only new points in this essay are: that gastric crises often occur without exacerbation of peripheral pains; that they may be divided into two forms, one of which is direct or central, the other reflex in origin. With the former there are many severe head symptoms, headache, vertigo, occasional diplopia colored vision, salivation, etc. The reflex attacks occur more suddenly, sometimes after meals, and are quickly and easily recovered from. The author admits lesions in the centres, and also in the peripheral nerves as the active causes of crises. Küsser,[230] of Halle, describes at length and treats of the physiology of the various symptoms, which are due to lesion of the vagi or their nuclei in the course of tabes.

Muscular Atrophy.—Remak[195] presented to the Neurological Society of Berlin a case of posterior spinal sclerosis complicated with atrophy of the abductor, opponens and flexor brevis pollicis, with partial anæsthesia of the distribution of the median nerve of the right hand. The upper extremities were not the seat of tabetic symptoms. The author considered the symptoms due to neuritis of the median nerve. The subject is generally examined and the views of other authors presented.

Arthropathy.—This subject was discussed in the Society for Internal Medicine at Berlin, by Bernhardt and Leyden.[194] Interesting cases were presented and related, but no new view proposed. Pierret[173] makes a preliminary and somewhat obscure communication upon the pareses which occur in the course of posterior spinal sclerosis. Some of them are due to peripheral neuritis, others (hemiparesis) to an encephalic lesion. A part of this paper can not be fully judged until the author's views are fully developed, but his claim that the overextension of the great toe in the ataxic walk is due to paresis of the flexor muscle, seems far-fetched and wholly theoretical. Overaction of the extensors and abductors in general go to make up the ataxic walk; and these aberrations of movement are, it seems to us, fully accounted for by impairment of muscular sense and a break in the apparatus for automatic spinal coördination. If Pierret's explanation of the movement is correct, we should apply the reasoning to the extravagant abduction of the larger members, which is observed.

Mollière[265] reports a case in which perforating ulcer of the foot and loss of teeth from the upper jaw were very early if not the earliest signs of tabes.

Etiology. — Teissier,[187] of Lyon, in a carefully elaborated lecture, opposes Fournier's view that syphilis is a direct competent cause of post-spinal sclerosis. He considers the etiology of this affection to be complex, and states that in order to obtain this lesion there must be (1) an inherited or acquired diathetic state predisposing to sclerosis (in general), and (2) a special exciting cause, inducing the location of the sclerosis upon the posterior columns. Under the first group of predisposing causes are: syphilis, gout, rheumatism, alcoholism, plumbism, and paludism. Exciting causes are: vertical coitus, injury to the spine, fatigue, direct inheritance of sclerosis and of neurotic disposition. In 35 cases these causes were represented as follows: predisposing causes: rheumatism, 14; positive syphilis, 13; alcoholism, 7; plumbism, 4; paludism, 2; gout, 2. Exciting causes: excessive (vertical) coitus, 18; traumatism, 5; excessive fatigue, 5; chilling, 2; neurotic heredity, 2; direct sclerotic heredity, 2. Karger[191] found certain evidences of syphilis in 53 per cent. of 117 cases. Nägeli[199] has collected 1403 cases of tabes from various authors, of which 46.1 per cent. had presented positive evidences of syphilis. Including suspected cases, the percentage rose to 60.6. On the other hand, among 1450 non-tabetic cases from various clinics, 9.5 per cent. had certainly had syphilis; and including suspected syphilis, the proportion increased to 22.2 per cent. Hutchinson[225] considers syphilis, fatigue and excessive sexual indulgence, acting upon a neurotic constitution, as the most important causes of the disease.

Pathology and Pathological Anatomy.—Lissauer[196] has studied by means of Weigert's method, the lesions of the posterior cornua in this disease, with respect to their chronology. This study is based upon the following analysis of the normal anatomy of the horns, which, according to the author, are composed of three parts: (1) a spongy zone in the substantia gelatinosa, contiguous to the marginal zone (inner margin), and in which the fibres of this zone are lost; (2) the substantia gelatinosa, strictly speaking, in which are found the large fibres from the posterior roots and of the lateral parts of the posterior columns; also fine fibres constituting the terminal network of the posterior spongy layer; (3) the spongy substance which extends from the substantia gelatinosa to the base of the horn, and which may further be divided into an anterior and posterior zone. The fine and large fibres of the posterior roots traverse this spongy area. The posterior horn therefore receives two sets of fibres from the posterior roots. (1) Large fibres which go directly into the spongy substance where they change directions. (2) Fine fibres, which immediately after the entrance of the root, deviates laterally and forms an ascending fasciculus, part of which ascends close to the lateral column, another is lost partly in the substantia gelatinosa and partly in the posterior columns. Specimens from five cases were examined with the following results: (1) the marginal zone (associated with the posterior radicular fibres) is almost invariably degenerated. In three cases there was here an independent area of degeneration. These are early changes. (2) The fine fibres of the posterior horn are less often and less severely affected; a length of time being required for the lesion to spread from the marginal to the deeper fibres. (3) The large radicular fibres are attacked at a relatively late period and degenerate slowly. (4) Clark's columns are always affected, and early in the course of the disease; at first the fine medial fibres derived from the lumbar region are affected, the lateral fibres and cells being diseased much later.

Takács[197] treats of the anatomical relation of the posterior columns, posterior horns and Clarke's columns in a complicated and evidently largely imaginative manner. Among other statements may be quoted that the columns of Goll and the ascending cerebellar fasciculi are made up of equivalent (?) fibres which are, through the cells of the posterior horns, a continuation of the posterior roots; and that the most direct sensory conduction takes place through these white columns. Yet he is puzzled to account for the rarity of lesion of the ascending cerebellar fasciculi in tabes. He admits (pathologically) two forms of tabes: (1) one in which the lesion is primarily in the posterior gray substance and its ganglion cells, and the white fibres of the posterior columns and roots are affected by secondary degeneration; (2) a form in which in posterior spinal meningitis is the initial change. These are very hazardous statements, but the final one, that the changes in the posterior columns are nearly always those of secondary degeneration, will appear still stranger to those who have studied the histology of the disease.

Oppenheim and Siemerling[200] have made an exhaustive histological study of the central and peripheral nerve lesions in 14 cases of progressive locomotor ataxia, and have also examined the peripheral nerves in a number of fatal cases of other diseases, as tuberculosis, alcoholism, marasmus, senility, lead paralysis, etc. The essay does not lend itself to brief analysis and must be read in the original by the special student. With respect to peripheral nerve lesions found in the latter class of cases, the authors rightly insist upon the fact first established by S. Mayer, that a few degenerated fibres are to be found in the nerves of healthy mammals and of man. In various pathological or toxic states they find more fibres degenerated than Mayer allows as normal, but no symptoms indicated the condition during life, and they conclude that a certain intensity as well as extension of nerve degeneration is required to cause recognizable symptoms (including DeR.). The frequency of pressure paralysis of the musculo-spiral nerves in alcoholized subjects they explain by supposing that prior to the pressure, the fibres of the nerve were unhealthy (*vide* lesions of nerves). With respect to the relation between symptoms and lesions in tabetic cases, they are unwilling to formulate any law as to which symptoms are of central and which of peripheral origin, although in many individual cases the problem may be solved by careful observation (including electrical tests). They consider Pitres and Vaillard's statements on this point as premature and unsound.

Treatment and Prognosis.—Suckling[177] has obtained relief of pain in three cases by the use of antipyrin in 10 grain doses. Teissier[187] admits the complete cure of certain cases by anti-syphilitic remedies and refers to one perfect result under the care of his father, B. Teissier. Raison[223] has successfully treated the pains of tabes by application of spray of ether and of chloride of methyl to the painful area, to the trunk of the nerve which supplies it, and also to that part of the spinal column which corresponds to the origin of the nerve (or its plexus). The author prefers ether to chloride of methyl. Hutchinson[225] believes that in many cases, if the patient will restrain his sexual indulgence and avoid fatigue, and if circumstances generally are favorable, the disease may be wholly arrested in its early stages; but the cases brought forward in his paper do not support this statement. Rest, an abstemious life, and anti-syphilitic treatment (when indicated) are his chief remedies. Babinski[226] related to the Biological Society three cases out of Charcot's private practice and hospital service in which a partial cure (arrest in one case and positive improvement in two cases) was obtained. In the arrested or abortive case the lesions of posterior spinal sclerosis and atrophy of the optic nerves were found after death. M. Benedikt[229] addressed a letter to the same society, in which he advanced rather rose-colored views of the curability of tabes. The communication is sharply critical of the predominant pessimistic views; it is general and does not give corroborative instances in detail. In 30 years' practice, the author has had "a very large number" of arrested and retarded cases. The types of tabes which offer a relatively good prognosis are: (1) Those with preceding optic nerve atrophy. (2) Those with preceding gastric crises. (3) Those in which the morbid process extends to other systems of the cord and to the brain (the favorable prognosis applying only to the tabetic symptoms). (4) The acute or subacute cases and (5) the syphilitic cases. Therapeutical measures are (1) careful, prolonged and repeated galvanic treatment, (2) absolute rest, wet cups to spine, ergot and nitrate of silver, the spinal ice bag (in the acute or subacute cases), (3) anti-syphilitic treatment, (4) nerve-stretching, which the author regards as the most important treatment in the future. Brown-Séquard,[235] while agreeing with most physicians as to the inutility of counter-irritation to the spine in this disease, recommends the systematic use of cautery points upon the lower extremities, producing a therapeutic effect by reflex and inhibitory action.

THE KNEE-JERK IN HEALTH AND DISEASE.

Lombard[152] has made an extensive study of this phenomenon in health and disease, and has greatly perfected the methods for developing and registering it. Two of the improvements are: the placing of the subject in the lateral decubitus at ease, with the lower extremity suspended and well balanced; this eliminates much of the unconscious muscular effort which the subject makes when examined in ordinary positions, and a greater amplitude of motion is obtained. The second improvement consists in developing the jerk by the blow of an automatically acting hammer, the force of the blow being subject to graduation, and the moment of impact registered upon the drum of the graphic apparatus by electric connection. The normal knee-jerk is as a rule least in the night, and greatest in the morning after breakfast. All conditions which lower the excitability of the brain and spinal cord cause a diminution of patellar reflex; while it is heightened by opposite conditions, such as wakefulness, food, bracing weather, etc. In the Chicago Medical Society[232] a paper was read and a discussion followed upon the absence of knee-jerk in health. Dr. Curtis claimed to have observed this absence of jerk in several persons, including himself, in whom there were no signs of spinal disease. The general opinion was that such absence was excessively rare, and had become more so since the introduction of Jendrassik's method of testing while the patient is exerting his will-power upon some other muscular group, such as the hand (Mitchell's reinforcement).

Westphal[35] reports a case of tabes with autopsy, in which the knee-jerk was absent on one side but present on the other. The sclerosis was found to be almost strictly one-sided, at the junction of the lumbar and dorsal cord. The same author [154] describes two cases of combined sclerosis in which the knee-jerk did not wholly disappear until shortly before death; in one case 22 days, in the other about 2 months. In a previous publication[231] he reported a case of sclerosis limited to portions of the posterior columns, in which the knee-jerk was at first increased, and remained demonstrable until death. The explanation of this persistence of the reflex is that a part of the posterior columns lying mediad of the posterior columns to an imaginary line drawn from where the substantia gelatinosa makes an angle inward, parallel to the median septum, to the periphery remains normal. Westphal calls this the root-entrance zone, but how it differs in topography from Charcot's "zone radiculaire" is not clear. Sclerosis of this area is held by Westphal to cause cessation of knee-jerk, and by Charcot to be the initial lesion of true tabes. Hirt[233] has also recorded three cases of posterior spinal sclerosis (one verified by autopsy), in which the patellar reflex was present.

COMBINED SPINAL SCLEROSIS.

Dana[163] read a very interesting analytical paper upon this subject before the New York Academy of Medicine, based upon the study of 16 selected cases from literature and 8 cases observed by himself. The chief result of the analysis is to show that lesions and symptoms appear in irregular order and present unequal degrees of development in different cases, rendering the clinical designations given to the disease (ataxic paraplegia by Gowers, progressive spastic ataxia by Dana) insufficient and to a certain extent misleading. As regards symptom-grouping, the cases were divisible into two classes: (1) cases in which the symptoms of posterior spinal sclerosis were complicated with a spastic or tetanoid condition of the legs; (2) cases in which a spastic paraplegia or paresis was complicated with pains and anæsthesia, indicating disease of the sensory division of the cord. The gait in some cases showed a mixture of ataxia with spastic weak movements. The lesions were found in the pyramidal tracts bilaterally, in the posterior columns, and in the ascending cerebellar tracts. In several cases, especially in the cervical region, the sclerosis extended well into the antero-lateral columns. This fact was held by Starr, in the discussion of the paper, to vitiate the term of fascicular sclerosis as applied to this affection. Seguin, in the course of the discussion, read brief notes of a case of the second class, and showed sections from the spinal cord at different levels. It was an example of Dana's second group, few sensory symptoms having occurred. Yet the lesion was largely developed in the posterior columns (not simply in the columns of Goll). There was extension of the sclerosis away from the pyramidal tracts so as to include considerable of the lateral columns, and at different levels this deviation from "strictly fascicular lesion" was variable. Westphal[154] relates two cases, illustrated by numerous drawings, of sclerosis of the posterior and lateral columns, in which during life the patellar reflex had been retained until a short time before death. A remarkable vaso-motor disturbance occurred in the first case, viz., cyanosis with œdema of the hands, and to a degree, of one foot.

FRIEDREICH'S DISEASE.

Under the misleading title of "Tabes Dorsalis in Childhood," Freyer[167] reports three cases of what probably was Friedreich's disease (although in one case the symptoms indicated involvement of the medulla and brain), in one family consisting of eleven children, of whom only two were healthy. Three are said to have had "scrofula," and one was born dead, so that a reasonable suspicion of inherited syphilis obtains. F. Mendel[214] reports the clinical features of a case of a child 4½ years old. Symptoms as usual in this disease, and sensibility preserved in all its modes. Nine children; the sixth died at 4½ years with similar symptoms; patient was the last child. The author follows Leyden in classing this affection as a "combined sclerosis." Under the title of hereditary ataxia, Mastin,[240] of Mobile, relates the very interesting history of a family in two generations of which all the males (whose mothers were healthy, yet were the means of transfer [atavism]) were affected, at ages varying from 10 to 15 years, with the following symptoms: weakness of the legs, and a staggering, incoördinate walk, saddle-back weakness and incoördination of arms and thighs. The patients observed by the author, three in number, out of six, rose from a sitting or crouching posture precisely as do patients affected with pseudo-hypertrophic paralysis. Sensory disturbance not observed in any case. The incoördination was not truly ataxic. These cases are, it seems to us, miscalled; they are cases of hereditary juvenile muscular atrophy, and in many respects the symptoms tally with those described by Erb. The attitudes, gait, coarse incoördination of hands, atrophy of homologous arm and thigh muscles, go to make up the picture. The spastic phenomena, the ataxia and the occasional anæsthesia of Friedreich's disease are wanting.

SPASTIC PARAPLEGIA.

Pierret[164] made a preliminary communication to the Société de Médecine de Lyon, in which he proposes to designate the affection as motor tabes, in contradistinction to sensory tabes. We must beg to protest against the introduction of the term tabes, revived from prepathological days. It is a meaningless term, and if progress has not yet enabled us to find the pathological name of a disease, the next best thing is to give it a semeiological or clinical designation which shall bring to the mind some notions of its chief symptoms. Major[179] and Rabagliati gave cases of infantile spastic paraplegia, with ordinary symptoms. The interest of the communication lies in the fact that in two cases the Achillis tendons were cut without any benefit. Charcot[238] states that the presence of spastic paraplegia should always suggest disseminated sclerosis.

Treatment.—Mitchell[209] in a letter on this subject recommends a combination of a special form of massage and tenotomy. The manipulations are based upon the physiological fact that stretching where the excitability of a muscle, each and every movement should be fully and slowly performed on every accessible muscular group, most effort being expended upon the over-active muscles. The author justly—in our opinion—condemns "braces" and circumcision.

AMYOTROPHIC LATERAL SCLEROSIS.

In a thesis on this subject, written under the inspiration of Charcot, Dr. Florand,[159] the author, has for his principal object, to oppose Leyden's idea that the symptom-group so ably described by Charcot and his pupils is a secondary or later manifestation of labio-glosso-laryngeal paralysis. Florand claims just the opposite, viz., that the bulbar symptoms are to be looked upon as the lesions of amyotrophic lateral sclerosis. Two points in favor of this view are interesting: (1) the fact that observation of pure labio-glosso-laryngeal paralysis have been very rare since the study of amyotrophic lateral sclerosis, and (2) that in a few cases of this disease, the disease has been traced beyond the pons along the pyramidal fasciculi to the motor cortex where the large ganglion cells were found reduced in number and diseased. (Köhler and Pick,[160] Kojewnikoff,[161] Charcot and Marie[162]). In some cases the contracture is very slight or fugitive. In others bulbar symptoms have not appeared when a complication kills the patient (Kojewnikoff's first case.) In other cases the symptoms of the bulbar lesion exist alone for a time. Among the exaggerated tendon-jerks, the new jaw-jerk is interesting. It may be developed by tapping the chin on either side of the median line, or depressing or tapping the lower incisor teeth. The author attaches no diagnostic value to electrical reactions (variable and usually not degenerative).

The diagnosis is usually easy in the typical form, but in others it may be most difficult to distinguish it from ordinary progressive muscular atrophy, disseminated sclerosis with contracture, and simple lateral sclerosis. In the matter of pathology, the author accepts Vulpian's conclusion that the "sclerosis" consists in a primary change in the nervous elements of the pyramidal tract,—a parenchymatous myelitis; but this is certainly premature. Many studies are yet needed to clear up the natural history and pathology of this affection. This thesis is marred by incomplete, misleading, and false bibliographical references. Lennmalm[189] reports a case in which the lesion of the pyramidal tract could be traced from the motor areas of the cortex downward along the well-known paths. Marie,[190] on the other hand, records an instance in which there was no trace of sclerosis at the level of the crura cerebri. Zacher[206] relates an instance of dementia paralytica in which amyotrophic lateral sclerosis symptoms appeared. After death, besides the lesions common to both diseases, the peripheral nerves were found extensively degenerated. The changes in the anterior horns were very slight.

PSEUDO-HYPERTROPHIC PARALYSIS.

Inglis[205] presented to the Detroit Medical Society four patients, members of one family, having the usual symptoms of this disease, and Emerson mentioned at the same meeting four other cases, likewise of one family in which mental disease was rampant. Raymond[207] gives a minute description—with plates—of two cases. The same cases had previously been made the subject of a communication by Bourdel. Another brother had been similarly affected, but there were no other cases in the family. The father had syphilis a few years before marriage, without later symptoms, but most of the 12 children of this marriage had died young. Portions of muscles were removed by Duchenne's trochar from the right quadriceps, ext. femoris, and from the left calf. The fibres were pale and somewhat smaller than normal; their striation was present, but difficult to see and in places almost lost. In some parts, the fibre was changed to a hyaline pale mass. Nowhere was fatty or granular degeneration found. The principal lesion was an increase of the connective tissue, with formation of fatty tissue, compressing the muscular fibres: a condition of sclero-lipomatosis. Jacoby[208] examined the muscles in a case of this disease in a boy æt. 15 years, and reached the conclusion that the disease was essentially a chronic inflammation invading both the perimysium and the muscle tissue. The process consisted of a gradual reduction of the muscular fibres into medullary or inflammatory corpuscles, which in turn went to form partly fibrous, partly cartilaginous and partly fatty connective tissue. He was uncertain whether the muscular or the connective tissue change was primary. He thought the pathological process was related to that which produces myositis ossficans progressiva, and would be inclined to call it myositis hyperplastica progressiva.

NORMAL TENDERNESS OF THE FOURTH AND FIFTH DORSAL VERTEBRÆ.

Harkin[237] renews his claim that these vertebræ are normally sensitive to pressure, and that the region over them is peculiarly qualified to receive therapeutic impressions (by blisters, etc.) for the cure of various neuroses.

Replying to a criticism that (as all well know) these vertebræ are not normally tender to reasonable pressure the author explains that in such cases, *i.e.*, where no pain is felt under moderate pressure, the physician must direct the patient's attention to the part and even tell him "I know better than you," and go over the spine again, tapping smartly with the knuckles (*sic*).

With this rough method and the suggestion, it is no wonder that the author has obtained confessions of pain from almost every one examined. Besides, the author naively infers that the tenderness found over the spinous processes corresponds to tenderness of the spinal cord beneath

HEMIATROPHY OF THE TONGUE.

Pel[210] reports the case of a probably syphilitic male, æt. 34 years, who developed the following unilateral symptoms: (1) Atrophy of the left half of the tongue, with protrusion to the left. (2) Paralysis of the left side of the soft palate. (3) Atrophy of the left sterno-mastoid and trapezius muscles. (4) Paralysis of the left vocal cord. No DeR. was present, only reduced excitability.

The author refers these symptoms to a lesion affecting the left nuclei of the spinal accessory and hypoglossal. In one respect this explanation seems insufficient, viz., as applied to the palatal paralysis. He states that this part is innervated by the "ramus internus of the vago-accesorius," but we would suggest that as two muscles of the palate, viz., the levator palatæ and azygos uvulæ are innervated by the facial nerve, through Meckel's ganglion and its palatal branches, that there was probably also a partial lesion of the left facial nucleus in the oblongata (the facial muscles were normal).

[This case was accidentally omitted from the section on lesion of the medulla oblongata.]

EPILEPSY.

Semeiology and Diagnosis.—Oliver [250] has made a critical examination of the eyes of 50 adult male epileptics, nearly all Americans. The principal abnormalities found were increased retinal vascularity and visibility of the perivascular lymph channels. There were in many cases heavy striations emanating from the disk, due to increase in thickness of the fibre-layer. Extra ocular motions intact in all cases, yet author admits weakness of the intern and explains it by presence of Hy. and Hy. As.,—results of no special value. Hystero-epilepsy has been studied in four boys by Laufenauer.[252] Besides character of attacks, other symptoms for differential diagnosis from common epilepsy are concentric reduction of visual fields, achromatopsia of various forms, the presence of tender or hysterogenous points, inequalities of pupils (left wider), greater loss of weight after attacks. Anæsthesia is rare in hysterical boys. Sexual irritation not mentioned. Lebrun[253] epileptiform attacks dependent (?) upon slow pulse. In July, after a miscarriage, the patient's pulse fell from 70 to 32 beats per minute. Two days later occurred a first syncopal and epileptiform seizure (clonic spasm after syncope); pulse 18. Afterward the pulse ranged from 25 to 40 beats, and five more attacks occurred, in the last of which the patient died. (October.) No autopsy. There were various cerebral symptoms before the pulse began to slow—vertigo, diplopia, epigastric distress, yellow vision, etc., and these recurred occasionally after. Most probably there was a bulbar lesion inhibiting the action of the vagi and leading to the various phenomena. Bourneville[254] has made a fresh study of the temperature in the paroxysmal period of epilepsy, and in contradiction of Witkowski[255] maintains his former statements (and Charcot's) that the epileptic attack is always followed by increased temperature. Several tables of observation on numerous (82) patients are given. His conclusion is that (1) isolated paroxysms of epilepsy increased the central temperature; (2) this increase varies from 0.1° C to 1.5° C and sometimes more, the average being 0.5° or 0.6°. The same author[280] publishes fresh data and diagrams of the temperature in fatal status epilepticus, supporting his former statements relative to the occurrence of pyrexia in this condition. A fatal case is fully reported, with a minute description of the brain. Bourneville[258] rehabilitates the epilepsia procursiva of older writers in an elaborate essay which has run through four numbers of Charcot's Archives, and is not yet completed. We cannot, consequently, give a full analysis of the paper, as the pathological anatomy and physiology of the symptom "procursion" have not yet been published. Procursive epilepsy is a variety characterized by attacks which consist in a rapid run in a straight line or in a circle, seldom followed by fall or by coma, but accompanied by congestion of the face. The run lasts about as long as an ordinary seizure. Cases presenting this symptom are classed as (1) E. procursiva strictly speaking, in which there are no other forms of seizure. (2) Ordinary E. with a procursive aura. (3) E. with procursion after paroxysms. (4) Procursive vertigo. In the fourth installment of the essay the number of cases described reaches 25. They are studied with that minuteness as regards heredity and etiology for which the author is distinguished, and most of them are completed by autopsy, in which the configuration of the brain (gyri and sulci especially) is carefully noted. These records will prove valuable for the study of epilepsy in general.

Psychic epilepsy, in the shape of attacks of mental disturbance presenting well-marked peculiarities, may occur, says Leidesdorf,[244] in persons who have ordinary epilepsy, and also in persons who have none (or none that can be demonstrated). In some cases an apparent substitution takes place and motor discharges are absent for a long time, while the mental disturbance prevails. Excitement and violence are characteristic of these paroxysms, as well as suddenness of onset, rapidity of maximum development and of cessation. Amnesia of what occurs in the attack is, says the author, a constant symptom. In cases where such or similar paroxysms occur in individuals who have never had common epilepsy, the diagnosis is often difficult. It is safe to consider them epileptic explosions only when the following factors can be determined: (1) etiological facts, such as neurotic heredity, alcoholism, great fright; (2) when a sort of aura exists before the paroxysms; (3) if the attacks arise suddenly to a maximum, rise rapidly and suddenly cease; (4) if complete amnesia exists. The medico-legal importance of these conclusions, backed by the author's great experience, is extreme. Bannister,[248] on the contrary, holds that in so-called epileptic mania (he refers more particularly to pre-epileptic and post-epileptic outbreaks) loss of consciousness is not a necessary factor. In the so-called automatic epileptic conditions, there may be a state of double consciousness, in which it cannot always be said that the mental functions in the abnormal condition are less perfect and complete than in the normal state. (But here amnesia exists—the two states are separated by an absolute break.)

Baker,[259] in a short but valuable communication, deals with the mental symptoms which occasionally develop when ordinary epileptic attacks are suspended by bromide treatment. He relates three cases of varying degree of psychic trouble which support his statement that in such cases the bromide treatment should be persevered in and combined with tonic treatment, life out of doors, and especially removal from home (though not to an insane asylum). He points out the necessity of carefully distinguishing between symptoms of bromism and truly maniacal disturbance. Harriet C. B. Alexander[260] deals with the same subject under the title of "Dangers of the Bromides in Epilepsy," but does not seem to discriminate between bromism and the substitutive delirium on the one hand, and the terminal condition of agitated or violent dementia which leads many epileptics to the insane asylum ultimately at the present time, as it did years before the bromides were known. (The same criticism applies to the alleged production of simple dementia by bromides.) The author is not prepared to advise the omission of the bromides and advocates a mixed treatment.

Pathological Anatomy.—In the Philadelphia Neurological Society, Dec. 27, 1886, Dr. Dercum[242] read a valuable study of 12 epileptic brains and a discussion by Mills and others followed. In all these brains abnormalities of sulci and gyri were found; in quite a number sclerosis of the skull had occurred. In some of the brains there was over-development of the occipital lobe, with abnormal sulci. In several the parieto-occipital and interparietal sulci were confluent, producing an ape-like arrangement. In a number of brains the sulcus of Rolando opened into the Sylvian fissure. Abnormal sulci were also observed in the frontal lobe. In two brains the cuneus, in another the lobus quadratus were enormous. One wholly pathological sulcus traversed the first temporal gyrus. The author classifies these abnormalities into (1) morphological and (2) pathological. In almost every instance it was evident that mechanical hindrances to the development of the brain had existed; but there had also been truly cerebral pathological processes at work. Bourneville and Baumgarten[243] report an extraordinary case of alcoholism in a rachitic child 5 years old, accompanied by epilepsy and dementia. Excessive use of white wine from the age of 3 to 4½ years. Was intoxicated the day before first convulsion. Autopsy revealed a soft condition of the brain, with decortication, dependent upon meningo-encephalitis. The cunei were very small (child partly blind during last few months). Histological examination revealed interstitial sclerosis and lesions of the pyramidal ganglion cells, most marked in the occipital cortex. Zohrab[247] claims to have found one lesion uniformly present in the brains of four epileptics of different ages (18 to 16 years), viz., softening of the subependymal white matter around the extremity of the posterior horn of the lateral ventricles. The lesion was most marked in the hemisphere opposite the most severe spasms. In all these cases, the Ammon's horns appeared normal to the naked eye. Pepper[278] made an autopsy of a man having chronic epilepsy with mental failure, in whom antifebrin controlled the attacks to a certain extent, when given in doses of 8 grains *ter die.* Death occurred from suddenly developed coma, with spasm. The autopsy was absolutely negative, as regards brain, heart and kidneys more especially. It is important to add that the antifebrin had been reduced to two doses a day before the coma, and the man's wife stated that he had had four other attacks of a similar nature. No description of the brain is given.

Pathology.—A careful résumé of the various experimental data upon which a theory of the epileptic attack (motor form) may be framed is by Vetter.[261] He concludes that in Jacksonian epilepsy the cortex is primarily affected (excited) while in idiopathic cases the course of the discharge is primarily in the white substance, with extension of the excitation to the adjacent cortex. Lemoine[261] gives two cases of epilepsy occurring in subjects having mitral regurgitation and one case with aortic regurgitation. In the first cases the symptoms were of grand and petit mal, preceded by a precordial or cardiac aura, and accompanied by cephalic congestion. In these cases, which the author believes to have been epilepsy from cerebral congestion, digitalis subdued the attacks after the bromides had failed. In the case with aortic disease, the symptoms were those of cerebral anæmia; caffein controlled the seizures. The author expressly states that he does not positively claim that there is a cardiac epilepsy. Vierra de Mello,[227] of Rio de Janeiro, Corresponding Editor of the ANNUAL, makes a short but vigorous plea in favor of the influence of hereditary syphilis in the genesis of epilepsy, hystero-epilepsy, and hysteria.

Etiology.—Bourneville and Baumgarten[243] (*vide supra*) record a case in which abuse of white wine, often to complete intoxication, in a child of 3 to 4 years old, developed meningo-encephalitis, causing many symptoms, prominent among which was epilepsy (grand mal and petit mal) and dementia. Marie[279] strongly combats the idea that hereditary influences (directly epileptic or neuropathic) is a strong or frequent cause of idiopathic epilepsy. He rightly insists that the epileptic attacks themselves constitute only a symptom or symptom-group, and that this must be due to some morbid condition or lesion. Nearly always, he thinks, the actual cause of epilepsy is post-natal in its occurrence and consists in a cerebral lesion or disease. Considering that the same brain lesion may exist in two children, one becoming epileptic and the other not, he is obliged to admit heredity as a predisposing cause. The whole argument is insufficiently elaborated, and the conclusions rather premature.

Treatment and Prognosis.—Thompson,[245] in an essay read before the New York Academy of Medicine, explained his method of treatment in detail. They are not based upon any attempt to determine the pathology of any individual case, especially as relates to the brain-condition, but more upon a consideration of the patient's general condition. He insists upon the primary necessity of improvement of nerve nutrition, for which purpose he recommends cod-liver oil, phosphorus, (at least he thinks he employs this agent when he gives phosphoric acid and hypophosphites); cold baths and prolonged sleep are not mentioned. He clings to the fast-waning fetish of abstinence from "all butcher meat" for a period of two years. The anti-epileptics employed are the bromides, with occasional addition of belladonna, oxide of zinc, chloral hydrate, Hoffman's anodyne, and when there is vascular disturbance and nocturnal enuresis, digitalis. A favorite remedy is a red pepper pack (ʒj to Oj of hot water). In many cases small doses of mercury are of much assistance.

Rockwell,[246] at the same meeting, read a paper upon the treatment of epilepsy by galvanization. He thought it a valuable agent, but did not claim that it alone could cure the disease. Its effects are specially marked in the nocturnal variety. The method of application should be central galvanization and general faradization (vide Beard and Rockwell's treatise). The treatment should be continued with suitable intermission for at least two years after the last occurrence of epileptic symptoms. The method, along with ordinary medicinal treatment, had been tried by him in 28 cases. In 10 the patients had given it up too soon to allow a judgment to be formed. In 3 cases, electricity seemed useless if not injurious. In 8 cases, a certain amount of benefit was obtained, and in 7 cases, great improvement, 2 of them recovering completely (the length of time from last seizure not stated). An obvious and practical objection to this treatment is that it requires daily or almost daily attendance on the physician's or patient's part for long periods of time, entailing expenses (if the application is made by one skilled in electro-therapeutics, as it should be), which exceedingly few patients can bear. It is almost like prescribing a trip to Europe to an overworked junior clerk. The measure may be beneficial, but its application restricted. Sinkler[249] reports the cure of a case by circumcision. A boy æt. 3½ years; first epileptic attack at 16 months, recurring with increasing frequency. No rachitis and none of the ordinary causes of epilepsy present. Penis very irritable and frequently handled; no phimosis. Bromide treatment did not diminish the fits but produced bromism. Circumcision done August 6, 1886. When examined December 27, no return of attacks had occurred. [Bromides had been continued at the rate of 15 grains per diem.] This case is without value as it was reported at too early a date; besides, the very early appearance of the first attack is rather against a sexual causation; irritability of the penis may well have been of central origin, and improvement obtained by traumatic inhibition. We request a further report of the case. Bennett and Gould[251] have successfully (?) trephined for epilepsy. A male æt. 36 had a first epileptic seizure six weeks after a severe contusion of the right side of the head with loss of consciousness. In the succeeding six years attacks occurred on the average once a week; many were followed by paroxysms of fury. General health remained good. There were no objective symptoms except a mobile cicatrix in the scalp above and behind the right ear, pressure upon which sometimes caused red light before the eyes and loss of consciousness. No true aura, but was irritable just before attack. All remedies proving useless, Gould trephined on July 8. The bone and dura were normal and several punctures in the brain revealed nothing. The wound healed readily, and when last seen, December 8, there had been no return of convulsions. This case also is prematurely reported, and should be made the object of a further statement.

Laufenauer[252] recommends for treatment of hystero-epilepsy in boys, isolation or removal from parental care, a general tonic treatment, and occupation. Shramm,[256] of Dresden, reports two cases cured by removal of ovaries. Both cases were clearly of menstrual epilepsy, in origin and periodicity; in both a strongly marked heredity was present. In the first case, four attacks occurred after the operation (normal ovaries), and at time of report, 15½ months after last seizure, there had been no relapse. In the second case (only small cysts in one ovary found), three seizures occurred after operation, then none for 10 months when last heard from. These cases are open to the objection already made, viz., too early a report. Hughes[257] writes a very optimistic article on the treatment of epilepsy chiefly by bromides and galvanism. Bromide of potassium, combined with Fowler's solution and syrup of the hypophosphites, in doses appropriate to the age, idiosyncrasy, etc., of the patients, is the mainstay. Other bromides may also be used. But it is not stated whether the remedy is to be given uniformly and continuously, or otherwise, or for how long a time. Other remedies are also employed according to minor indications, and strychnia is rejected as contra-indicated. Great stress is laid upon cephalic galvanization, antero-posteriorly, and from either side to the opposite hand, with a mild current, done daily for several months. Regulation of habits and hygiene generally is insisted upon. The author's hopefulness may be judged by the expression, that if the physician carries out such a treatment conscientiously, under favorable circumstances, he will "have for his reward more successes than failures." Would it were so! This claim is in part accounted for by the author's statement that in many cases 18 months or 2 years is the length of the treatment. Yet how many patients relapse in the third year, yea, even in the fourth, who have given up the bromides after a two years' or three years' limit. Our limit, in the most favorable cases, is four years after the last epileptic attack of the least sort. Spitzka[262] bases his treatment on the following view of the pathology of the disease. "Epilepsy is a diseased state of the encephalon, without a palpable characteristic lesion, and manifesting itself in explosive activity of an unduly irritable vaso-motor centre, leading to complete or partial loss of consciousness, which may be preceded or followed by various phenomena expressing the undue preponderance of some, and the suspended inhibitory influence of other cerebral districts." "To give the bromides alone is to postpone the explosion and generally intensify it," is a statement which will hardly meet with general approval, though it may be true of a few cases. He does give the bromides, however, and prefers that of sodium. One large dose is better than three smaller ones. Ergot he has found of decided benefit. Conium has been especially valuable in controlling motor and mental symptoms: dose, from 15 to 60 minims of Squibb's fluid extract, not over three times a day. Solution of glonoin (nitro-glycerine) 1 per cent. in doses of from one drop upward, does good service. The state of the abdominal organs should receive especial attention, because of the intimate relation of the pneumogastric nerves of the vaso-motor centres. Good hygiene should be insisted upon. Very correctly the author insists that the drugs employed in treating a case of epilepsy should not be stopped suddenly, but decreased. We regret that the author does not give us his limit of treatment after the last attack.

Franks [266] makes a preliminary report on the case of a man who injured the frontal and parietal bone on the left side in 1879. The injury was followed by unconsciousness lasting three weeks, and apparent complete recovery. In 1883, convulsive attacks began and lasted continued at varying intervals. Trephining done Feb. 22, 1887, revealed a cyst the size of a walnut, containing an old clot and serum, in a depression of the brain. In 12 days the wound had healed, and at time of report in June, no attacks had occurred. Schufeldt[268] publishes (prematurely also) a case of supposed traumatic epilepsy in which trephining was done. Male æt. 40 years, injured on top of head, on left parietal region, one half inch from median line, some 30 years before. Had no symptoms until March, 1887, when he had a first spasm followed by another in about three weeks. In 1872 he had contracted constitutional syphilis, from which he had suffered more or less until 1884, when he went to Hot Springs, Arkansas, and had a long-continued treatment. When seen by Schufeldt he had impairment of memory, unequal pupils and hesitating speech. On May 6 trephining was done, but the bone and dura presented no evident lesion. Two months later no attacks had occurred. This case was evidently one of chronic cerebral syphilis (pseudo-general paresis) with epileptic seizures, and was entirely unsuited for operation. A case in which the operation resulted fatally is reported by Wheeler.[269] Houzé[271] operated on a case of hemi-epilepsy caused by injury to the left parietal bone; the spasms always began on the right side, then passed over to the left; there were also ataxic aphasia and lingual hemiparesis. In three years frequent attacks, greatly controlled in 1881 by large doses of iodide of potassium and a cervical seton. Operation, January 25, 1887, revealed a splinter of the inner table with a whitish appearance of the dura (not opened). Rapid healing, followed by a return of all the symptoms in February, when the treatment was resumed as above. No fit occurred till May 4, then none till time of discharge. This case will hardly go on record as a "cure." The cortex was very probably the seat of disease.

Antifebrine (acetanilide) was given to 11 epileptics by Salm[263] in Jolly's clinic at Strassburg, in doses of from 15 to 45 grains *per diem.* The result was unsatisfactory; in no case were the paroxysms suspended, while in 7 cases the attacks became more frequent. Nearly all the patients developed cyanosis. Leidy, Jr.,[264] has employed both antipyrine and antifebrine in various nervous affections. Of 26 cases of epilepsy, 14 were greatly benefited by acetanilide (gr. iij two and three times a day), and 3 by antipyrin (grs. x to xv three times a day). Cases of petit mal proved more amenable to treatment. Hare[270] derived benefit from antifebrine in three cases of chronic epilepsy. Da Costa[272] had used bromide of nickel in doses of one grain several times a day, according to tolerance (in pillular form or solution). Frothingham[276] reports a case (female æt. 24), in which epileptiform attacks preceded by photophobia and flickering had existed from the eleventh year until the spring of 1884, when he discovered a total hypermetropia (masked by spasms of accommodation), and gave 1.50 D. glasses. From report by parents, no epileptic attacks occurred after this to February of this year (date of report). It is a pity that the attacks are not more exactly described, and that nothing is said of petit mal and other epileptic symptoms, family history, etc., for the case is suggestive of hystero-epilepsy rather than of true epilepsy. Bridges[274] argues that epileptics should not use flesh foods, even meat soups being objectionable. Dana[275] expresses much more rational and, in our experience, much more practical views when he states that the diet should be adjusted to the patient and not to the disease. Meat he considers harmless in most cases, and positively beneficial in lithæmic and anæmic cases. Fatty foods, and particularly milk, are valuable. He advocates small meals, and if necessary, more than three a day; no heavy meal to be taken within four hours of bed-time.

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PERIPHERAL NERVOUS DISEASES AND GENERAL NEUROSES.

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MULTIPLE NEURITIS.

MULTIPLE NEURITIS, or polyneuritis, is a disease in which many nerves are attacked by inflammation at the same time, the inflammation having a tendency to affect the nerves in a more or less uniform manner, as will appear later when we come to discuss the pathology and symptomatology of the disease. The affection is sometimes spoken of as multiple "peripheral" neuritis; but the use of the word "peripheral" is unnecessary. All nerves are peripheral, and such an expression means nothing unless it is intended to refer to the nerves after their exit from the skull and spinal canal.

According to Buzzard,[1] a description of typical multiple neuritis, observed in Paris, was given forty years ago by Graves, who also declared that the French pathologists sought anxiously but in vain to find the cause of the strange disease in the nervous centres; and Starr tells us that an American physician, Dr. James Jackson, of Boston, in 1822, painted a graphic picture of one of the forms of this affection. From time to time cases of the disease have been reported under various names, and papers upon the subject have been written by Magnus Huss, Duchenne, Duménil, Leudet, Pitres, Jaccoud, Eichorst, Joffroy, Leyden, Lancereaux, Grainger Stewart, Roth, and others. The last three years, however, have been a season of great increase in our exact knowledge of the subject, and medical literature during this time has teemed with fruitful observations and investigations. The year 1887 shows an abundant literature; but while many contributions have appeared in various languages, a few are pre-eminent because of their completeness or their originality. First among these stands the work of an American neurologist,—the *Middleton-Goldsmith Lectures on Multiple Neuritis and its Relations to Certain Peripheral Neuroses*,[2] by Dr. M. Allen Starr, which lectures were delivered under the direction of the New York Pathological Society, and have since been published in various medical journals. They constitute an exhaustive study of the subject up to the date of their delivery, and reflect the greatest credit on their author. By Dr. J. B. Duplaix,[3] the subject has been reviewed in a masterly manner; but, strange to say, we find scarcely any allusion in his paper to American work, although French, German, English and other European authorities are copiously mentioned. Many valuable articles and notes of cases or groups of cases have also appeared in different languages,—among others, in America by Chapin,[4] Dana,[5] Biggs,[6] and Folsom;[7] in England by Ross,[8] Clarke,[9] Edwards,[10] and Fawsitt;[11] in Germany by Thomsen,[12] Kast,[13] and Laquer;[14] in France by Pitres and Vaillard,[15] and others.

Beri-beri, commonly regarded as an endemic multiple neuritis, has also had, during 1887, a copious and interesting literature, at the head of which is an able monograph by Weintraub of Holland. This literature will be more fully stated and discussed when beri-beri is especially considered.

Pathology.—Theoretically, in considering the pathology of neuritis, it is possible to recognize several forms; thus, we may have (1) a perineuritis, the inflammation affecting solely or principally the sheath of the nerve; (2) a parenchymatous or degenerative neuritis, the nerve fibres themselves being involved; (3) an interstitial neuritis, in which the connective tissue between the nerve fibres is chiefly attacked; (4) a diffused neuritis in which all parts of the nerve bundle are more or less affected. The clinical distinction between perineuritis and the other forms of neuritis has not yet been clearly made. Possibly it is asking too much to attempt such a differentiation. The two forms of neuritis which seem to be universally recognized are the parenchymatous and the interstitial. The microscopical appearances observed in these forms of inflammation are well described by Starr, who also gives, by way of introduction to the pathology of multiple neuritis, an interesting account of the histology of nerve fibres, and also of the changes resulting from nerve degeneration artificially produced in animals.

Parenchymatous inflammation is probably simply degenerative. The myelin is absorbed and eventually the axis cylinder disappears, and finally only an atrophied tube deprived of its original contents remains.

With reference to this parenchymatous inflammation, it is important that the appearances shown are identical with those seen in secondary degeneration of nerves after diseases of the spinal ganglion-cells, or after compression or local disease of the nerve trunks. This fact has led Erb,[16] and we think with much show of reason, to argue that the nerve changes in this form of so-called multiple neuritis are really secondary to a primary affection of spinal trophic centres. Starr agrees with Strümpell and others, in opposing this view of Erb, but Erb's hypothesis seems to us to have much in its favor. Starr holds that against the view that the nerve changes are secondary, are the facts that the degeneration is usually limited to the distal portion of the nerves; that the sensory nerves are affected, centrifugal degeneration of these nerves from spinal lesion being unknown; and, that a parenchymatous neuritis has its parallel in other parenchymatous inflammations and primary scleroses. The one argument of the three which seems to us to have any weight is that which relates to the inflammation attacking sensory afferent as well as motor efferent nerves. We believe that eventually at least some of the cases which are now classed as multiple neuritis of the parenchymatous variety will be relegated to the spinal cord; whether all such cases will be is, however, doubtful.

As to the forms of multiple neuritis which are classed as interstitial or diffused, little or no doubt can exist that they are true nerve inflammations. Here are to be seen inflammatory changes of the endoneurium and perineurium, with formation of new connective tissue, and destruction by compression of nerve fibres. "In this class," says Starr, "the mere inspection shows the nerve to have been the seat of pathological changes, for it is either congested, swollen, and lacking in lustre, or it is yellow and irregularly swelled by the accumulation of fat, or it is evidently reduced to a mere connective tissue strand."

Gombault[17] has described a special form of neuritis, the so-called *segmental periaxillary neuritis.* It is important to notice this variety, as it is present in toxic neuritis from lead poisoning, diphtheria, alcohol, and other forms of nerve inflammation with which we are concerned in the present paper. By inducing chronic lead poisoning in pigs, Gombault produced a parenchymatous neuritis, in which a segment lying between the two nodes of Ranvier was diseased, while the other sides of the nodes remained healthy. In two cases of alcoholic paralysis, according to Clarke, this condition was found by Drs. Dreschfeld and Grainger Stewart. Pitres and Vaillard have noticed a somewhat similar condition in the neuritis occurring after diphtheria. Hadden, Eichorst and other observers have found the change to be an interstitial neuritis.

Etiology and Varieties.—A study of the clinical varieties of multiple neuritis is really an investigation into causation, as the only available practical classification of the disease is etiological. Following Starr, Duplaix, and others, multiple neuritis can be divided into (1) *spontaneous* or *idiopathic;* (2) *toxic;* (3) *infectious;* (4) *epidemic* or *endemic* (*beri-beri* or *kakké*). Beri-beri may be placed under the infectious variety; but it has so much special importance as a tropical or sub-tropical epidemic or endemic disease, that we prefer to treat of it in a special chapter.

(1) Under the name of *spontaneous* or *idiopathic multiple neuritis*, are placed cases which may occur anywhere from uncertain or unknown cause, although they frequently appear to be due to great fatigue, exposure to cold, damp or wet; or to mental excitement, or over-exertion combined with fatigue. A few interesting cases of this kind are cited from various authors by Starr and Duplaix. Starr calls attention to one practical point in connection with them, namely, that in some the early pains were in the joints and led to the diagnosis of rheumatism.

(2) Under *toxic multiple neuritis*, are included cases due to alcohol or other narcotics and stimulants, to lead, arsenic, copper, carbon bisulphide, carbonic oxide, ergot, etc. Besides these, according to Duplaix, in auto-intoxications, and in certain morbid states such as jaundice and uræmia, localized paralyses occur; but it is uncertain how far we have neuritis in such cases, the cerebrospinal apparatus in most of them being undoubtedly involved. While it might be highly interesting, space will not permit us to cite cases of multiple neuritis due to toxic substances. We will content ourselves with saying that numerous cases are now on record, in which autopsies and microscopical examinations have

demonstrated the existence of multiple neuritis in cases of paralysis due to the toxic agents above mentioned.

Several cases have been added during the present year. An interesting and valuable case of alcoholic multiple neuritis is that reported by Biggs, who presented the spinal cord and nerves from a case of alcoholic paralysis at the Meeting of the New York Neurological Society, March 1, 1887.

The autopsy showed that the patient was greatly emaciated. The legs and thighs were markedly flexed. The muscles of the legs were of a yellow color, and apparently converted almost entirely into fat. The muscles of the thighs were less affected. The spinal cord, nerve roots and trunks were normal in appearance. Microscopical examination showed the spinal cord apparently normal, with the exception of a slight sclerosis in the columns of Goll in the cervical region. The nerve roots were normal. In one of the sacral nerves before its exit from the spinal canal was found a marked increase in the endoneurium with diminution in the number of the nerve fibres, and an irregularity and indistinctness in the appearances. The right sciatic nerve showed the same changes more marked. In the posterior tibial the process was even more advanced, and in this only an occasional nerve fibre could be detected. Microscopically the gastrocnemius was composed almost entirely of adipose tissue; only here and there atrophied muscle fibres were found. The small nerve trunks in the muscle showed advanced degenerative neuritis, with comparatively little new growth of connective tissue in the nerves.

(3) *Infectious multiple neuritis* includes those cases which follow or are complicated with simple pharyngitis, diphtheria, scarlet fever, rötheln, measles, small-pox, typhoid and typhus fevers, tuberculosis, epidemic cerebro-spinal meningitis, leprosy, mumps, cholera, syphilis, or puerperal infection. Rheumatism, gout, diabetes, and probably some other diseases, whether recognized as infectious or not, are associated with or give rise to multiple neuritis.

The reported cases with autopsies and microscopical examinations, which show that of the various forms of paralysis following such diseases as diphtheria, scarlet fever, measles, small-pox, typhoid, typhus, and malarial fevers, some at least, are due to neuritis, either local or multiple, are gradually increasing. Starr and Duplaix record a considerable number of these cases, which we can only allude to in the present connection. We will more specifically discuss some of the other forms referred to under the above heading, although we cannot go to that length in the subject which its importance demands. Kast reports the case of a girl who had an attack of follicular sore throat, which he distinctly states was not diphtheritic. It was followed by paresis of accommodation, progressive atrophy of the arms and legs, disturbance of all forms of sensibility, with retardation of conduction of pain, but never any tenderness on pressure in the nerve trunks. Wasting paralysis of the interossei and the tongue, with abolition of faradic contractility, and feeble response to strong currents of galvanism, were also noted. The knee-jerks were abolished. The symptoms of bulbar paralysis were also present, as paralysis of the velum palati, disturbance of the innervation of the larynx, and loss of power in swallowing. The patient died in nine months. The post-mortem showed the central organs healthy, and degeneration of the cerebral and spinal nerves.

According to Gowers,[18] the nervous symptoms of leprosy depend almost exclusively on a neuritis which is typically interstitial. Danielssen and Boeck, and later Virchow, have shown the existence of neuritis or perineuritis, which explains the anæsthesias, the ulcerations, and the gangrenes observed in this disease. Several histologists have verified the facts advanced by the first observers. The muscular wasting, anæsthesias, etc., are greatest in degree toward the ends of the limbs.

Joffroy[19] reports a case of multiple neuritis following mumps. The paralysis affected all four limbs; the legs, as usual, were first affected; lancinating pains preceded the paralysis; the deep reflexes were abolished; the muscles gave the reaction of degeneration, and slight pressure caused pain. The paralysis endured for four months, and got well under iodide of potassium.

Duplaix states that in cholera limited paralyses are not rare, and that they are generally accompanied by troubles of sensibility. The clinical aspect in this disease is peculiar, seeming to indicate, according to Landouzy, that these phenomena are caused less by central lesions than by peripheral lesions attacking at the same time the sensory nerves as well as the motor. The analogy of the nature of different pyrexias and infectious maladies makes it probable that neuritis has existed before, but has not been perceived.

The occurrence of multiple neuritis in cases of tuberculosis is of great interest. In the wards of the Philadelphia Hospital, where tuberculosis in some form invades almost every department, we have frequently witnessed evidences of the probability of this complication. Starr refers to cases of multiple neuritis reported by Joffroy, Eisenlohr, Strümpell, Webber, and Müller, in which the patient died of phthisis; also to cases of Oppenheim in which multiple neuritis was present in tubercular patients but went on to recovery.

We have not seen any reference to the occurrence of multiple neuritis in the course of cerebro-spinal fever or epidemic cerebro-spinal meningitis. That it does occur either as a part of the disease or as a complication, can scarcely be doubted. During the month of February, 1888, in consultation with Dr. J.W. Dick, of Philadelphia, Dr. Mills has seen a case in which the symptoms of multiple neuritis seem to be combined with those of cerebro-spinal meningitis, also two similar cases with Dr. W. C. Cahall.

At the American Neurological Association, 1887, Mills[20] presented notes of three cases with the typical symptoms of multiple neuritis, in which the history, or the results of treatment, or both, indicated a syphilitic origin of the disease. With reference to alcohol, he said that the cases of the same class clearly due to alcohol alone, presented almost identically the same clinical features as those shown by purely syphilitic cases. Purely alcoholic cases did not, however, respond so quickly to specific treatment, and sometimes would not respond at all to such treatment. During the last ten years he had seen at least a dozen cases similar to those detailed, the majority of which had recovered under specific treatment with the help of massage, electricity, tonics and time. Duplaix and others have shown that the nerves are frequently affected by syphilis; but in the majority of cases it is not with a neuritis that the clinician has to do, but with gummatous neoplasms, or with the lesions which result from compression by such neoplasms; nevertheless incontrovertible facts point to the existence of syphilitic neuritis. If paralyses of the cranial nerves, and particularly the motor nerves of the eye, are the consequence of such lesion, it may be that the same is true in some cases of so-called sciatica, and painful affections of other large nerves.

Kast records a case of infectious puerperal neuritis, septic endocarditis commencing on the eleventh day after confinement. Forty-five days later the patient complained of numbness in the left hand and the next day of complete anæsthesia of both hands. Examination showed atrophic paresis in part of the territory of the ulnar and median nerves, with reaction of degeneration, smarting pains in both arms, the nerve trunks sensitive to pressure, paræsthesia and weakness in the legs. The patient recovered.

Rheumatism, says Duplaix, certainly sometimes causes congestive or inflammatory lesions of nerves. Neuralgia is a general feature in such cases. M. Besnier, according to the same author, has remarked the rarity of paralysis, but neverthless it is not exceptional to encounter it, for all the peripheral nerves may be attacked by the inflammation. Sciatica is very frequent and variable in its course. Facial neuralgias are not rare. Curious instances of peripheral paralysis in connection with articular rheumatism have been observed. A nerve like any other organ, may be under the influence of rheumatism before the articulations. Duplaix cites the case of a soldier who was taken with fever, vertigo, pains at the nape of the neck, and at the end of four days showed symptoms of paralysis of the right oculo-motor nerve, with retinal congestion. Towards the seventh day rheumatism of the left knee appeared, which spread to the right shoulder and to the feet. During this time the paralysis diminished and disappeared at the end of eleven days, while the acute attack of rheumatism lasted four weeks.

Pitres and Vaillard[21] have made some interesting investigations on peripheral neuritis in chronic rheumatism. Having had occasion to examine several peripheral nerves belonging to two patients subject to chronic rheumatism, they were astonished to find profound degenerations. On these facts, which they presented to the Society of Biology, they based the view of the possible coexistence of neuritis with chronic rheumatism. Their observations seem to prove that the peripheral nerves are often the seat of important degenerations in chronic rheumatics, since in the only three autopsies that they had occasion to make, they found traces, more or less deep, of these degenerations. The cord, it is true, was not healthy in any of the cases. Twice they found diffuse sclerosis and once posterior spinal meningitis; but they believe it would be altogether illogical to consider the lesions observed in these cases as the consequence of medullary or meningeal lesions, since in two of the cases where the rachidian roots were examined they were found to be normal.

Graves held that the sciatic, facial and other severe neuralgias often met with in youth were to be traced to the changes in the blood made by uric acid, which in its turn caused a congestion or inflammation of the nerves. Garrod believed that both the neuralgias and certain partial paralyses observed among gouty patients were probably dependent on a neuritis. Duplaix holds that sciatic neuritis is not rare among gouty subjects.

In diabetes, paralysis and painful affections of the nerves are common. Duplaix, for example, enumerates paralysis of the tongue, of the muscles of the eye, and one or more of the extensor muscles. These nervous troubles are transitory, while their rapid cure agrees ill with the idea of neuritis. Other much severer neuralgias—of the sciatic, trigeminal, intercostal and even pneumogastric—in diabetic subjects point more decidedly to an inflammatory affection of the nerves. We have treated successfully with sodium salicylate, blisters, and galvanism, a case of severe brachial neuritis occurring in a diabetic patient.

Symptomatology.—Multiple neuritis of whatever form, presents certain characteristic symptoms,—sensory, motor, vaso-motor, trophic, or special; and therefore we will be able to present a summary of the symptomatology of this disease which, with unimportant modification, will answer for any of its varieties.

In the first place, however, it must be remembered that whatever the variety, cases differ somewhat in their symptomatology, according to the acuteness and rapidity of the manifestations. In certain of the reported cases, the disease has had an acute and rapidly progressive course. Eichhorst, cited by Starr, Duplaix, and others, published a case under the title of *acute progressive neuritis.* The patient, a woman 66 years old, suddenly presented the symptoms of an acute inflammation of the left peroneal nerve; soon the other leg was involved, then the upper extremities, and finally the optic and pneumogastric nerves. In addition to the pain at the beginning, the symptoms were motor paralysis, anæsthesia, and abolition of the reflexes and electro-contractility. In six weeks the patient was dead. Duplaix also records other similar cases, among them an observation of Roth. In this case the disease began with dysphagia, and was rapidly followed by paralysis of the tongue, aphonia, paræsthesia, and paresis of the four extremities. The knee-jerk was abolished, and in six days the patient died from respiratory and cardiac involvement.

Strümpell cautions against certain cases of multiple neuritis of this kind being confounded with Landry's disease. Eisenlohr also in an autopsy of a case of ascending acute paralysis, found multiple neuritis. In these forms of polyneuritis the patients die in consequence of the invasion of the bulbar nerves.

Sometimes the course of the affection is so rapid as to have received the name of apoplectiform, as in an observation by Vierordt, reported by Duplaix. The phenomena presented are those of other cases of fatal multiple neuritis appearing with great rapidity, and accompanied by serious general depression, fever, thirst, headache, extreme lassitude, and even delirium. Albuminuria, congestion of the kidneys, and articular swelling with great pain, are sometimes present. The functions of the bladder and rectum nearly always remain intact. Such cases end ordinarily in death in a few hours, by extension of the paralysis to the muscles of respiration.

Mills has seen several cases of the acute rapidly progressive form of polyneuritis. In one, a young girl, the diagnosis of hysteria was made, the patient dying in about a week, having had the sensory, motor and other symptoms of multiple neuritis, and finally succumbing to paralysis of respiration. In another case the patient died after a painful illness of several weeks, the symptoms in general being those of a rapidly ascending paralysis. In this case several diagnoses were made by different practitioners, among others, acute locomotor ataxia, and anterior polio-myelitis. In a third case all the bulbar nerves were attacked in rapid succession, paralysis of the extremities occurring at nearly the same time, and the entire duration of the disease being only a few days.

Special causes to some extent impress peculiar features upon the symptomatology of multiple neuritis. In alcoholic multiple neuritis, for instance, the patient frequently has a precedent history of gastric catarrh, tremor, sleeplessness, and erratic pains; sometimes of numbness and psychical disturbances. Not infrequently, as will be hereafter more fully shown, alcoholic cases present delusional insanity as a prominent complication. In lead neuritis the presence of a blue line or blue discoloration of the gums, and the tendency of the paralysis to attack certain muscular groups with greater virulence, others escaping partly or entirely, serves to give a special character to the cases. The tendency of the supinator muscle to be attacked last or not at all, and when attacked to regain its function first as the patient recovers, is a notable peculiarity. Cases due to arsenic, mercury, copper, bisulphide of carbon, etc., may or may not have special peculiarities dependent upon the effects of the poison upon other organs and parts. In multiple neuritis following diphtheria, scarlet fever, measles, and rötheln, the tendency to involvement of the muscles of the eye, palate, pharynx, larynx, with or without more general paralysis, is well known.

The symptoms of multiple neuritis, whatever may be its cause and variety, are both subjective and objective.

Among the subjective symptoms nearly always present are pains and abnormal sensations referred to various nerve distributions. The patients use various terms in describing their sufferings, as numbness, tingling, burning, boring, tearing, pulling or stretching; less frequently they speak of darting, shooting or lancinating pains. The pains may be referred to nerves, to muscles, or to joints. Cramplike feelings may be present, as may also girdle, drawing, or constriction sensations. Sometimes coldness is complained of, limited, it may be, to islands or regions. Even sensations similar to those described by ataxic patients may be present; for example, the patients may complain of standing on wool. Heaviness in the limbs may be described before any objective paralysis is observable. Dimness of sight, impairment of hearing, or rarely perversion of smell and taste are occasional early subjective symptoms. Among symptoms which may be regarded as partly subjective and partly objective, and which are sometimes present, are initial fever with its accompanying constitutional disturbances, such as rigors and delirium.

The objective symptoms of multiple neuritis may be classed as sensory, including those connected with the special senses; motor, including paralytic, ataxic, and spasmodic phenomena, and alterations in electrical reactions; reflex, vaso-motor and trophic.

The objective sensory symptoms are among the most important from a diagnostic point of view. Tenderness of both nerves and muscles is a constant and distinctive symptom. "Hyperasthesia of touch," says Starr, "and also to electricity, is not infrequently observed during the first weeks. It is usually followed by anæsthesia, although this rarely becomes complete. In some cases the loss of tactile sense is quite evident from the onset, either limited to the cutaneous distribution of some special nerve, in which case oddly shaped areas of insensibility will be found, or, as is most often the case, about uniformly distributed over the distal parts of the extremities. When the anæsthesia is at its height, the patient has difficulty in locating a touch upon the limb, even though he feels it. The transmission of pain and temperature sensations is always delayed but the impressions are usually felt quite acutely. The sense of pressure has been tested only in a few cases, and in those it was decidedly impaired. The muscular sense escapes any affection in some cases, but in others is the most profoundly disturbed of all the senses."

Few investigations have been made into the conditions of the special senses in multiple neuritis. According to Starr, optic neuritis has occurred in several cases, and hearing as well as sight has been affected. Gowers makes no mention of opthalmoscopic appearances. In the report of Strachan, to be given later, the changes in the fundus revealed by the opthalmoscope, were varying degrees of retinal hyperæmia, according to the severity or duration of the disease, rarely amounting to optic neuritis. Strachan had not seeen optic atrophy, but there was generally an increase in the density of the pigmentation of the fundus. In very rare cases even smell and taste have been affected.

Motor paralysis is, of course, a prominent symptom in multiple neuritis. Its distribution and progress vary somewhat, but in a large number of cases a certain uniformity is observable; a marked tendency, for example, to early and decided inflammation of the musculo-spiral nerves of the upper extremities, and of the anterior tibial nerves of the lower; hence, dropped wrist and dropped foot are notable appearances. The palsy sometimes begins as a slight weakness or paresis, and increases either gradually or rapidly. In severe cases almost all the nerves of the legs and arms become involved, but the paralysis is nearly always more severe below the elbow and below the knees. Sometimes the muscles above these joints are palsied and even those of the trunk and the cranial nerves become involved; such cases closely resemble Landry's paralysis, and the worst forms of generalized anterior poliomyelitis. The paralyzed muscles relax and atrophy.

Changes in the electrical reactions of nerve and muscle occur often with rapidity, but owing to the hyperæsthesia it is often difficult to make a thorough investigation. The electrical responses vary with the amount of inflammation and degeneration in the nerves, all the way from a simple diminution of excitability to the faradic and galvanic currents, to entire loss of faradic response and galvanic reactions of degeneration. Buzzard justly insists upon the importance of a thorough examination of the electrical reactions in all cases where the presence of neuritis must be kept in view, and he shows by several instances the diagnostic value of this investigation. Kast attaches great importance to electric reactions as a means of distinguishing between poliomyelitis and multiple neuritis. When the same nerve gives different reactions with different muscles, the probable conclusion is that various branches of the nerve are variously affected; that in fact there is a multiple neuritis. Lloyd[22] believes that some assistance is given in diagnosis by a study of the electrical condition of the nerve trunks and muscles in neuritis and anterior poliomyelitis. The point which he emphasizes is the retention of electro-excitability in the nerves, which in chronic cases does not appear consistent with the idea of neuritis, but rather of a slow cord-lesion. If this electric excitability can be preserved in a chronic neuritis, we must suppose a very slow interstitial inflammatory process, which compresses some fibres and allows others to escape, which is highly improbable. Starr, in a discussion at the American Neurological Association, in 1887, disagreed with Lloyd. He had taken pains in many cases to make careful electrical observations, but had not been able to verify the position taken by Lloyd. Probably the correct position is that of Kast, and also that the electrical reactions assist in separating cases of multiple neuritis from forms of chronic non-inflammatory degeneration of the anterior columns.

The question of the involvement of the bladder and bowels is one of considerable importance. Starr looked upon the absence of any interference of the automatic acts controlled by the sphincters as a negative symptom of some importance. Certainly, as a rule, the bladder, rectum and sphincters are not involved in any paralytic or spasmodic disorder. Duplaix cites some exceptions to the rule that the functions of the bladder and rectum always remain normal,—a case of Pitres, and one of Francotte, as well as several recorded by Leyden, Grocco, and Eisenlohr. The bladder and rectum have their nerve distribution, and theoretically we see no reason why these nerves may not be attacked by multiple neuritis. As a matter of experience, they are very seldom affected.

With or without motor weakness, but usually with paresis, not seldom a distinct incoördination is observable. This is so prominent in some cases that the patients have been regarded as suffering from forms of locomotor ataxia, or pseudo tabes. At the meeting of the American Neurological Association, in 1886, Dana[23] read a paper on *Pseudo Tabes from Arsenical Poisoning*, in which he presented two somewhat unique cases of arsenical poisoning presenting to a large degree the appearance of tabes. Cases of pseudo tabes or ataxia from alcohol, lead, and other toxic infectious agents have been reported.

General convulsions are rare in multiple neuritis and perhaps are only present as a complication. In a case reported by Mills[24] as one of subacute generalized myelitis of the anterior horns, but which with newer light he is now inclined to regard as an example either of multiple neuritis or of multiple neuritis combined with polimyelitis, the patient was taken with spasms, light at first, but gradually increasing in strength. They lasted two weeks, and then ceased for a week, when one came on just after taking a hypodermic injection to relieve pain. This was the last spasm she ever had, as well as the most severe. Her nurse said that she was totally unconscious of it, and that her feet were drawn almost to the back of her head, her mouth being also drawn to one side. After that her feet and limbs began to pain her and to be very sensitive to the touch, the pain gradually increasing until the agony became almost indescribable, especially in both great toes, "as if some one was forcing a large pin under the nail and tearing it from the flesh."

Local tonic spasms, cramps, or contractures are comparatively common objective symptoms. The claw-shaped hand, and the equino-varus foot from spasm as well as from palsy, are frequently seen. The limbs may be contractured into various positions, flexion at the knees being frequent.

The skin reflexes may be either excited or depressed, probably according to the extent, severity and degree of the inflammation or consequent nerve degeneration.

The so-called myotatic irritability is usually lost early. We do not know of any differential studies as to the condition of knee-jerk and muscle-jerk. According to the experience of nearly all who have written on this subject, the knee-jerk is soon abolished. Some cases have been reported, however, with retained or even increased knee-jerk. Strümpell and Möbius,[25] for instance, from some, according to them, undoubted cases of multiple neuritis, controvert the assertion that the tendon reflexes are diminished or absent. In their cases the response from the tendons of the muscles of the thigh, arm, and shoulder blade, was exceedingly active. On curing the nerve inflammation the reflexes of the affected muscles became less intense.

Vaso-motor changes are present varying from a slight flushing to a deep purple; from slight pallor to lividity; from a scarcely appreciable swelling to a positive œdema. Even effusion into joints has been observed, but it is doubtful, according to Gowers, if this was the effect of the neuritis or was rheumatic and primary. Sometimes the glossy skin is present. Profuse sweating is occasionally observed. According to Starr, certain forms of atrophic disturbance are rarely met with in multiple neuritis, as for instance, ulceration, bed-sores, gangrene, and various eruptions. We believe, however, that these atrophic disorders are often present but have been overlooked or omitted in the reports of the cases. Raynaud's disease has been recently ascribed by Wigglesworth[26] to peripheral neuritis.

Complications.—The question of the complication of multiple neuritis with other pathological conditions, is one of great importance and to some extent has attracted the attention of observers and investigators. Mills, at a meeting of the Philadelphia Neurological Society, November 22, 1886, called attention to the concurrence of multiple neuritis with myelitis or encephalitis, holding that while cases of multiple neuritis pure and simple occurred, they were rare: and that multiple neuritis might be found associated with myelitis and encephalitis or both, or that it might coexist with meningo-myelitis, or meningo-encephalitis. While subsequent experience and investigation has led him to believe that pure multiple neuritis may not be so rare an affection as he at one time supposed, he is still convinced of the frequent concurrence of this disease with other affections of the cerebro-spinal axis. Some new experiences with such diseases as infantile paralysis and cerebro-spinal meningitis, have particularly confirmed him in these conclusions. Leyden held that in atrophic paralysis neuritis may coincide with the spinal cord lesion. Starr admits that no small difficulty may be encountered in settling the question whether in a given case we have to deal with multiple neuritis alone or with multiple neuritis which is complicated with myelitis. The number of autopsies in which this complication has been demonstrated, although few, are sufficient to make a determination of the question necessary. Starr gives some points which he thinks may enable this determination to be reached. These are based chiefly on the tendency of multiple neuritis to spontaneous recovery. According to Starr, the course of a case of multiple neuritis is quite characteristic, the symptoms reaching their maximum in a short time and then subsiding.

Mills, in the communication referred to on the concurrence of multiple neuritis with spinal and cerebral disease, called attention particularly to certain alcoholic cases with positive evidences of cerebral and spinal, as well as neural involvements. He has since made a number of observations on cases of alcoholic cerebro-spinal disease in which multiple neuritis is one factor. These cases may be divided into several clinical groups, at least three of which can be differentiated: (1) Mild cases which are not taken off their feet, and in which the probable existence of slight multiple neuritis is evidenced by numbness and pain in the extremities. In these cases the patient may show other evidences of chronic alcoholism in weakness of the limbs, lassitude, gastro-intestinal catarrh, etc. (2) Cases of well marked multiple neuritis of the type which has been described, namely, cases with marked paralysis, sensory disorders, vaso-motor and trophic disturbances, etc. In some of these cases myelitis may be conjoined with the neuritis, but brain symptoms are not present. (3) Cases of well marked multiple neuritis, with also undoubted cerebral disturbances.

Of this last class, Mills had seen several examples during the past year, nearly all of them beer-drinking women. He had seen two such cases in consultation with Dr. L. E. Taubel, of Philadelphia, from whom he has received some notes of which the following is a summary: Case 1, a married women 30 years old, was a steady beer drinker. She had suffered with gastric symptoms and had an attack diagnosticated as acute gastritis, the chief symptoms being nausea, vomiting and pains at the pit of the stomach. After five or six weeks she developed tingling and numbness in the feet and hands, loss of power below the knees, and slight paresis below the elbows. She suffered from irregular paræsthesia and anæsthesia; and at places intense hyperæsthesia, cutaneous and muscular. The main nerve trunks from the thighs down were also very tender to pressure. The bladder and bowels were not involved. The knee-jerks were absent. Her chief mental symptoms were difficulty in recognizing faces and remembering facts; she would frequently break into hysterical sobbing, and had a staring, dazed manner. She gradually recovered under sodium salicylate, iron, tonics, rest, good food, and massage. Case 2, a married woman, 60 years old, had for years taken beer in large quantities and was also in the habit of taking other alcoholic liquors in the morning. Among the earliest symptoms of her attack were severe pains about the ankles which had been diagnosticated as rheumatism. At the height of her trouble the symptoms were almost complete paralysis of the lower and upper extremities, with extreme hyperæsthesia over wide areas, with also irregular anæsthesia, absent knee-jerk, and other symptoms pointing to multiple neuritis probably conjoined with myelitis. The patient was evidently in a delusional state, and at times indulged in great screams and in singing; her memory was irregular; at times she remembered nothing; at others she recalled some things. Her paralysis and other symptoms deepened; she finally lost control of the bowels and bladder, and died in about six weeks after the beginning of her illness.

Cases have been reported which show that in locomotor ataxia multiple neuritis is a true complication.

Pitres and Vaillard[27] conclude, (1) Peripheral nerves of tabetic patients are often the seat of neuritis. (2) The neuritis of tabetic patients does not differ in any essential respect from the other forms of the non-traumatic affection. (3) The topographical distribution is variable, for the neuritis may attack the sensory, mixed and visceral nerves. (4) In the majority of cases, but not always, the disease begins in the outer extremity of the nerves. (5) The extent and gravity of the neuritis have no constant relation in respect to age, or the extension or depth of the medullary regions of the locomotor ataxia. (6) It is probable that the neuritis does not play any part in the production of the specific symptoms of tabes, such as lightning pains, incoördination of movement, abolition of knee-jerk, disorders of the muscular sense, etc. These depend upon the affection rather of the posterior columns of the cord. (7) Certain inconstant, added or complicating symptoms, however, appear to depend upon the neuritis; such, for example, as anæsthetic spots in the skin, localized trophic disease, localized motor paralysis, accompanied or not by muscular atrophy, isolated joint affections, visceral crises, etc. Duplaix cites also the researches of Pierret, Dejerine, Sakaky, and Oppenheim, as showing the importance of the changes in the peripheral nerves in the symptomatology of tabes. Strümpell[28] reports transitory paralysis of the radial nerve in a case of tabes,—a man aged 55, who was suddenly taken with paralysis of the left hand while reading his paper; all the muscles of the forearm were weakened by the radial paralysis.

In other forms of sclerosis, neuritis is also sometimes a complication.

Some of the forms of infantile paralysis are probably cases of multiple neuritis. These, according to Clarke, probably include all the cases in which recovery is almost complete, even after paralysis and atrophy have lasted a long time, while in the cases due to poliomyelitis, there is no recovery after the disease has lasted a short time. Chapin has reported cases of infantile paralysis in which were present wrist and ankle drop, with other characteristic symptoms of multiple neuritis. In one case both the cord and the nerve were diseased. He states that infantile paralysis due to anterior poliomyelitis generally occurs before the age of five, and nearly always before nine years; and lays stress on the sudden onset, regressive character of the paralysis, with absence of pain, in contradistinction to the ascending progressive palsy due to neuritis. Chronic anterior poliomyelitis, these authors say, is distinguished by the absence of sensory disturbance, and the order in which the muscles are involved, that is, in the ascending form, small muscles of the hand before extension of the fingers and forearm; in the descending, the gluteal muscles and those supplied by the obdurator and anterior crural nerves before the extensors of the legs. We have seen some cases of undoubted spinal infantile paralysis in which the paralyzed limbs were markedly tender to touch and pressure; but in these cases a neuritis may have complicated the myelitis, which was the chief affection.

Diagnosis.—Among the diseases of the spinal cord which closely resemble multiple neuritis, the most important are diffused myelitis, anterior poliomyelitis, and posterior spinal sclerosis; among other affections necessary to differentiate may be mentioned Landry's paralysis and hysterical paralysis. In diffused myelitis the symptoms are very similar to those presented by multiple neuritis; but in neuritis the functions of micturition and defecation are seldom affected, girdle sensation is rare, and the advance of the paralysis is usually from the legs to the forearms, the thighs and trunk escaping. Other points are also given by Starr. From anterior poliomyelitis, the chief points of distinction relate to the presence and persistence of the sensory manifestations in multiple neuritis. The distribution of the paralysis in symmetrically situated muscles, says Starr, especially if these muscles are supplied by single nerves, and the further extension to muscles in other nerve domains, rather than the affection simultaneously of muscles which are grouped physiologically, that is, act together to perform one function, will point to neuritis. From locomotor ataxia, multiple neuritis differs in many respects, but the character of the gait is sufficient, according to Ross, to distinguish it. In neuritis there is a peculiar elevation of the knee in walking, with a drooping of the toes, and an unusual exposure of the sole of the foot to one standing behind the patient. Such a case is unable to elevate the toes if sitting on a chair with the soles of the feet flat on the floor. From Landry's paralysis, it differs in exhibiting the reaction of degeneration, with marked sensory disturbances and wasting of the muscles. From hysterical paralysis, for which multiple neuritis has been mistaken, a careful study of the case will be all that is required. The changed electrical reactions, absent or altered knee-jerk, and peculiar sensory symptoms, will easily enable one with any understanding of the subject to decide.

Prognosis.—Starr says: "The prognosis in multiple neuritis is good, provided the exciting cause can be removed. The only cases which form an exception to the rule are those whose constitutions are much impaired by excesses or other diseases; those who have so far indulged in alcohol, or are so completely soaked with arsenic or lead as to be unable to throw off the poison; and those in whom the disease begins with great suddenness, advances rapidly, and involves the phrenic and pneumogastric nerves. These cases die either of respiratory paralysis or of some complication. When a case has reached the stationary period, the prognosis is generally favorable, and if the encouraging signs of recovery already mentioned begin to appear, a cure may be promised. The possibility of the complication of myelitis must not, however, be overlooked, and if it occurs the prognosis becomes at at once unfavorable."

Treatment.—In the early stages of multiple neuritis, salicin, salicylic acid, sodium salicylate, salol, or oil of gaultheria, may be given combined with bromides; morphia or other anodynes may also be necessary. Either hot or cold applications, or both alternately, may be found of great advantage. Syphilitic cases should be treated with mercury and iodides; malarial cases with quinine and arsenic; lead cases with potassium iodide or ammonium chloride, etc. Alcohol should be strictly prohibited. In the chronic stage strychnia and other tonics may be employed, with warm baths and douches, massage, passive, duplicated, active, systematized active movements, and electricity. The galvanic current is to be preferred for regenerating the diseased nerves; but faradism may also be used, especially after some improvement has occurred.

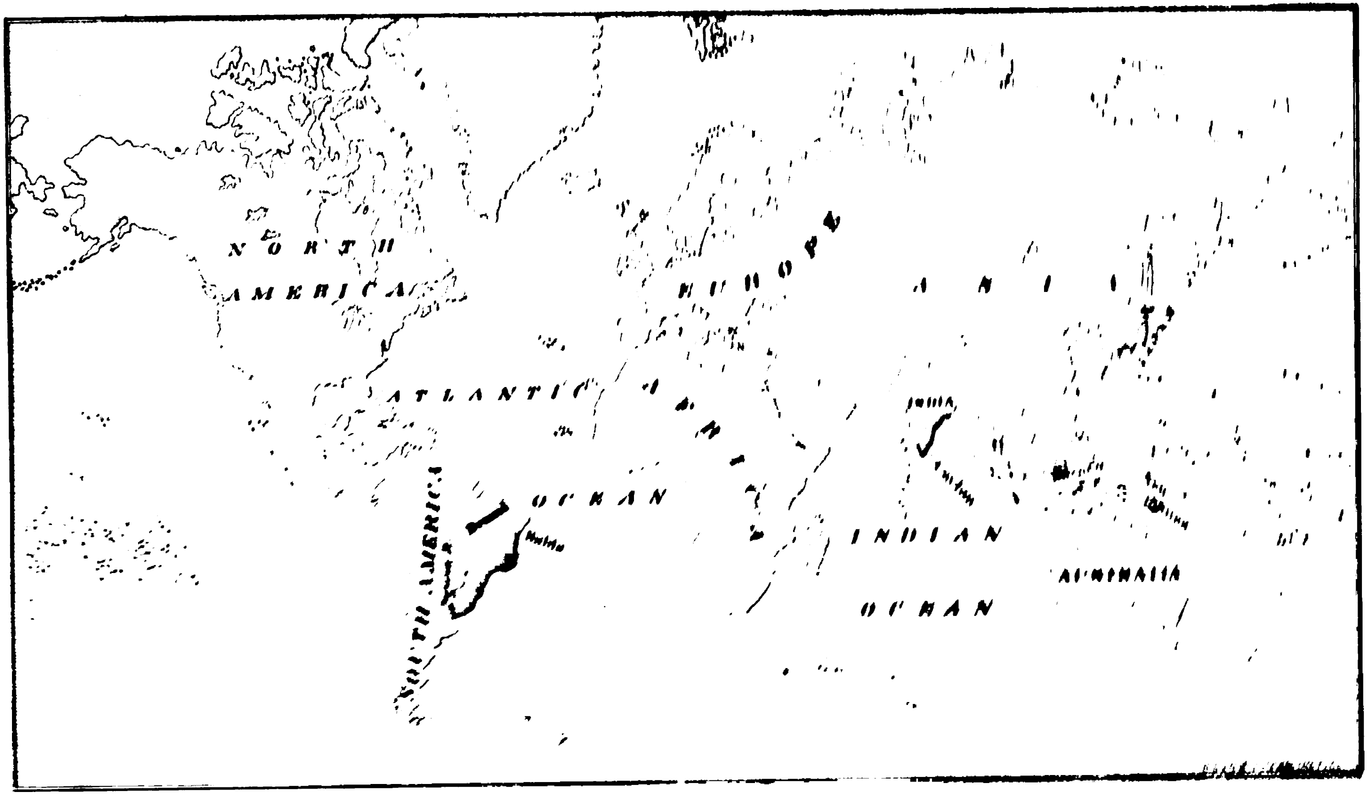
Semmola[29] reports a case of rheumatic neuritis or myositis, in which the use of pilocarpine was followed by an almost immediate cure, by its diaphoretic effect. The patient was a woman who had exposed herself, while overheated, to the draught from an open window. Violent pain and stiffness came on in the arm and shoulder. Passive movement was not impaired. Salicylic acid, electricity, massage and quinine had failed to give relief. Semmola gave two injections daily of ⅓ centigram of muriate of pilocarpine. After the second injection great improvement occurred and in four days the patient was cured. This remedy might prove of value in some cases of multiple neuritis.

BERI-BERI.

Starr supplies some highly interesting details concerning beri-beri, but the most exhaustive contribution on the subject is by Dr. Karl Weintraub,[30] Sanitary Chief of the Netherland East Indian Army. Like the lectures of Starr on the general subject of multiple neuritis, this series of papers leaves little to be learned from other sources, and to it we have resorted for much of the material of the present paper. During 1887, the French Government sent out Prof. Pekelharing[31] to their East Indian possessions, to investigate the nature and character of the disease under consideration. Cornelissen and Sugenaya[32] have also contributed a scientific report from the Dutch Indies. Van den Driessche[33] addressed a communication on beri-beri to the King of the Belgians, on which M. Barella presented a report, reviewing the entire subject, to the Belgian Royal Academy of Medicine. K. Takaki,[34] Surgeon-General of the Japanese Navy, and Prof. Baelz, of the Imperial Japanese University of Tokio, the latter one of the corresponding editors of the ANNUAL, have added facts and advanced views of great interest. From another of the corresponding editors of the ANNUAL, Mr. Henry Strachan, of Kingston, Jamaica, we have received a valuable original article on *Malarial* Multiple Peripheral Neuritis, a summary of which will be given later.

From Seguin,[35] of New York, the year has afforded some excellent notes on three cases of beri-beri, and some analogous indigenous cases of multiple neuritis; from Vineberg,[36] some clinical observations of an endemic of beri-beri among the Chinese coolies at the Sandwich Islands; from Marie[37] an account of the paralysis of the Isthmus of Panama, a form of beri-beri; from Roosevelt,[38] two papers on cases observed in New York; from Shattuck,[39] Stevens,[40] Pomroy,[41] reports of interesting cases; from Slater and Oliver,[42] notes of an outbreak of beri-beri on the Chinese transport ship, "Too Nan," stationed at Newcastle-upon-Tyne.

In the U. S. Marine Hospital Report for 1881 (referred to by Starr) is an interesting account by Dr. Hebersmith of the development of beri-beri in a Brazilian naval vessel, and in connection with the Report is given a map which we reproduce. It demonstrates the localities in which beri-beri is endemic.



MAP INDICATING THE GEOGRAPHICAL DISTRIBUTION OF BERI BERI

[illegible]

The derivation of the word beri-beri, according to Weintraub, is doubtful, and is given in several ways. Bontius knew the affection by this name in the 17th century. It is variously claimed to relate to the gait, *i.e.*, the Arabic word *biri* signifying "sheep," because of the way the animal walks, or from *biribi*, of the Malay-Japanese dialect, meaning "stiff gait." The disease has different names in the various parts of the Indian Archipelago. Van den Driessche, quoted by M. Barella, says the words *demi*, "half," and *ri*, "rice," indicate the fruit of the rice before it has attained maturity and that in writing the word "demi-riz," the medical service of the Netherland-Indies wrote the word "beri," and thence came the double word "beri-beri." According to Herklots, the word comes from the Hindostanee, *bharbari*, signifying "œdema" or "swelling." Carter says it is a corruption of two Arabian words, *buhr*, signifying "oppression" or "asthma," and *bahri*, rignifying "marine;" thus we have "buhr-bahri." Marshall says *bhayree* signifies in Ceylon "weakness," and the redoubling of bhayree (beri-beri) expresses a degree of extreme feebleness. By the Japanese the disease is called *kakke*, from two Chinese words,—*kiaku*, "legs," and *ke*, "disease." It has been known among the Chinese by this name for centuries.

Attention is called by the *Lancet* for August, 1887, to the fact that writers seem to have overlooked the very interesting observations of de Lacerda, of Rio Janeiro, who, in 1885, published a pamphlet, in Portuguese, entitled, *Hip Epidemic, or Marajo Epizootia; its Analogies with Beri-Beri*, in which he gives an account of the fatal disease affecting horses and swine in the Island of Marajo. This affection appeared to be due to a polymorpheous microphyte belonging to the ascomycetes, which abounded in the water of Lake Arary on the Island. This microphyte was found to produce the disease in a number of animals,—rabbits, pigs, birds, monkeys, etc., inoculated with it. The symptoms and the lesions found in the spinal cord after death, corresponded very closely, in de Lacerda's opinion, with those of beri-beri, and he came to the conclusion that the Marajo epizootic was identical with that disease. If so, his researches throw considerable light on its etiology, and are certainly well worth the study of those interested in the subject.

Dr. H. H. McCandliss, Corresponding Editor of the ANNUAL, from the Island of Hainan, China, writes that he has a specimen of a fossil crab found in the south of the island. It has a great local reputation as a cure for glandular swellings, and more especially for *beri-beri.*

Pathology.—We will give interesting results of numerous autopsies in cases of beri-beri as collected by Weintraub (condensed somewhat from the account given in the SATELLITE for February, 1888). The brain and cord and their membranes were normal, but the cerebro-spinal fluid had a slight reddish tinge, explained by the terrible death struggle. The *peripheral nerves were normal.* Hydro-thorax, pulmonary œdema and congestion were present in proportion as the disease was of the hydropic variety. Sometimes there were sub-pericardial ecchymotic striæ. In the majority of instances the heart was so much enlarged in all directions, and so markedly hypertrophied, as to have been called "cor borinum." In 50 autopsies of Weiss and Lodewyk, 47 showed forms of hypertrophy. Well marked atheroma of the large arteries was almost invariably present. The interna of the small vessels was always normal. The blood was hydræmic, its corpuscles diminished in number, and its fatty constituents increased. Haga found the leucocytes increased in number. Usually more or less dropsy of the peritoneum was present, and sometimes it was excessive. The stomach was distended, and its mucous lining often ecchymotic in places; the liver, spleen and kidneys were generally hyperæmic, but not structurally changed; the intestines were normal, except an occasional congestion and a slight ecchymosis of the mucous membrane. Erni found large number of trichocephalus dispar in the gut, while Stammesbaur, on the other hand, found nothing but anchylostoma duodenali in almost all cases. Lodewyks and Haga both agree that all striated muscles underwent fatty degeneration.

The post-mortem appearances are inadequate to explain its symptomatology. Weintraub holds that the appearances of the heart are secondary. The cause of the disturbance of circulation is in the disease of the vessel-walls, especially in the larger arteries. The atheroma of the great vessels indicates furious inflammatory drain, lessened calibre of the vessels, consequent strain and dilatation of the left heart, and secondary involvement of the right, with venous stasis, œdema, defective oxidation and dyspnœa. He believes that the bacillus of beri-beri is taken in through the lungs and carried to the heart and great vessels. He joins Lodewyks in believing the motor and sensory disturbances to be spinal, because of the symmetrical involvement of muscle groups. Scheube and Bælz are in favor of ascribing these disturbances to peripheral nervous disease. Schneider and Simmons explain the motor disturbances on a myopathic basis, but it has not yet been determined whether the muscles are primary or secondary in degeneration.

Tscholowski, of St. Petersburg, who examined several cases of this disease post-mortem, found well marked degenerative atrophy of peripheral nerves, with slight atrophy of some ganglion cells in the lumbar, which latter alteration he was inclined to consider secondary occurrence. Others have made similar reports as to the nerves.

Etiology.—Gayet, quoted by Barella, holds that the causes of beri-beri are: (1) Defective nourishment; (2) damp cold and the variations of temperature in a tropical country; (3) the depressing influence of a hot climate on the vaso-motor centres; (4) a series of secondary causes, as the influence of former maladies, especially diarrhœas and dysentery, anæmia, nostalgia, and bad hygiene.

Takaki succeeded in obtaining permission from the Japanese Admiralty, in 1883, to make a number of experiments in the service. He found that the food taken by men attacked by kakke was very deficient in nitrogen and contained a great excess of carbon, their relative proportion being twenty-eight to one or more. Observations were made by him on a voyage of the Riujo, a training ship. Prof. Baelz, of the Imperial Japanese University, of Tokio, Japan, Corresponding Editor of the ANNUAL says that Takaki now states in the Annual Report of the Health of the Japanese Navy, 1886, that a great improvement has taken place as to the occurrence of the disease in the Navy. It has almost disappeared. Takaki attributes the improvement to the change of diet—bread, wheat, and beans having been partly substituted for rice.

Dr. Henry Lanning, correspondent at Osaka, Japan, writes that in treating kakke (beri-beri), whatever may have been their notions of pathology, many of the native physicians appear to have found out the value of changing the diet of the patients,—prohibiting rice, and substituting beans, barley, etc.

Ogata, of Japan, and de Lacerda, of Brazil, claim priority in the discovery of a bacillus peculiar to beri-beri. Wallace Taylor's investigations also appear to show the existence of a bacillus in the blood. (Report of Dr. Henry Lanning, Corr. Ed.)

According to Pekelharing,[43] beri-beri is so much allied to infectious diseases that it seems reasonable to look for its cause in the lower organisms that invade the body. He found bacteria in the blood in all cases at Atchin. Beri-beri is commonly met with in fixed places and buildings, especially where a large number of people are congregated, as for instance in jails and barracks, and even in ships delayed on the coast. He does not consider that there is sufficient evidence for the opinion that the cause lies in certain kinds of food. It cannot be considered as a purely infectious disease, since no fresh case occurred among the out-patients in the neighborhood, when there were twelve patients suffering from the disease in the Hospital. It appears probable that it is transportable, but does not always find a congenial soil. The clear increase of the disease about a month after the overflowing of the river encourages the belief that the inundation had a decided influenee. The improvement of the circumstances consequent on stringent sanitary measures and disinfectant precautions, at the end of the year was rather marked. If the disease is due to septic organisms which germinate in the earth, and in dwellings where large numbers of people are congregated, Pekelharing believes that by constant cleansing and disinfecting, it would be possible to combat it, if not wholly, yet partially. He believes there is ground for hope in the future that the soil may be rid by degrees of the poison. That beri-beri is an infectious disease, Weintraub thinks hardly admits of doubt, and many authors are agreed upon this point.

Symptomatology.—Weintraub distinguishes between two varieties of beri-beri, the *paralytic* and the *hydropic* or *œdematous*, and says that there is sometimes a *hydropic-paralytic*, or combination form. Ninety per cent. of these cases, according to Van Leent and Weintraub, are of the hydropic variety. Some claim a *polysarcose form*, but Weintraub and others show the increased thickness of the fat layer to be due to the inhibition of serous fluid by the fat cells. Beri-beri may also be either *acute* or *chronic*. In the former variety death results in several days or a few hours. The chronic form admits of recovery, though this is often prevented by dysentery and febrile complications.

In the *hydropic* form, the patients are usually well nourished, have œdema of the lower limbs, anæmic mucous membrane, especially the conjunctival, lividity of the lips. The œdema begins on the back of the foot, at the ankles, and extends to the legs and thighs. In rare instances the scrotum becomes so œdematous as to attain the size of a child's head, a fact only mentioned by Weintraub. Van Leent states that in complete paralysis the œdema extends to the foreskin, belly and chest wall, neck, face and upper limbs. In marked cases, the patients with difficulty lift the lower limb at the knee, the foot, however, remaining on the ground, or only raised by a sudden genuflexion and lifting with the hands. When later the foot is allowed to fall to the ground, the toes touch first, which is a pathognomonic sign of beri-beri, according to Weintraub. As a rule, no effusion, or very little, takes place in the serous cavities in the first stages, and if this happens, the prognosis is unfavorable. In the advanced stage of hydrothorax, dyspnœa, cyanosis, dulness, and disappearance of the respiratory murmur occur. The heart is considerably affected. The pulse is at first weak and regular, but increases in frequency until the augmenting cardiac weakness and effusion into the serous cavities make it irregular, and sometimes intermittent. Simmons and Van Leent mention severe vomiting as an ominous symptom of the last stages. Others have never observed it. The urine is diminished, of a dark color and rich in urates. Occasionally there is retention with marked hydrops. There is never uræmia. Opinions differ as to albuminuria. It is probably rare. Urinary and fæcal incontinence are rare. Schutte is the only one claiming that retinal hæmorrhages occur. The motor and sensory disturbances may develop suddenly or gradually. The gait is very much like that of locomotor ataxia. Plantar sensibility is normal. The paresis of the lower limbs may end either gradually or suddenly in complete paralysis. Lodewyks, Furnée, and others, have noticed progressive involvement of the upper limbs and scapular muscles; the former has seen laryngeal paralysis.

The motor symptoms are generally preceded by sensory disturbances, such as creeping sensations in the lower limbs, and pain in the calves of the legs due to muscular cramp; marked paræsthesia, and hyperæsthesia, followed by marked anæsthesia. The temperature sense is undisturbed. The knee-jerk is usually totally lost. Weintraub says the death agony is most terrible to witness. The signs are all those of suffocation. Respirations frequently take place after the heart has ceased to act. Most authors consider pulmonary œdema and heart paralysis as the causes of death.

Electrical excitability of the muscles is decidedly lowered in the first stages, and totally lost with the onset of paralysis. Baelz, corresponding editor, reports that Takaki found almost no contraction on indirect galvanization; direct galvanization of the muscles showed the degeneration of Erb, generally incomplete. The contractions were much slower than in the normal state. These results corroborate those obtained by Baelz in 1881.

Diagnosis.—The diagnosis of beri-beri is comparatively easy in an endemic locality, particularly in its well marked stage. The prodromal period may be confounded with other diseases, and manifests itself in tiredness of the legs, indisposition and inability to work, depression, etc.

Prognosis.—The prognosis of beri-beri, according to Weintraub, is always uncertain and in acute cases invariably unfavorable. According to Lodewyks, there is a better chance of recovery in the paralytic form because there is less danger of sudden death from dropsy, pulmonary œdema, or cardiac paralysis. Recovery is tedious but may take place even after far advanced paralysis; but it is necessary for the patient to leave the locality where the disease is endemic. Convalescence continues from four months to more than a year. Relapses are not uncommon.

Treatment.—Wernich, according to Weintraub, recommended puncturing the skin and tapping to relieve hydrops, but such measures are not to be recommended. Weiss, Lodewyks, and others, recommend the muriate of pilocarpine subcutaneously, the latter claiming for it marked diuretic as well as diagnostic properties. Digitalis, because of the degenerated heart muscle, must be given cautiously for the relief of the palpitation. Claret and cognac are given to stimulate and assist digestion. As long as the patient is able, moderate outdoor exercise is urged, and after that electrical stimulation of the muscles. In the death agony stimulants are given. More important than all is the removal of the patient quickly from the endemic region to a mountainous district where the disease has never existed. Prophylaxis is the only way in which to meet this disease with success.

MALARIAL MULTIPLE PERIPHERAL NEURITIS.

Dr. Henry Strachan, Corresponding Editor, sends a communication on this affection as observed by him in 510 cases treated in the Kingston Public Hospital, Jamaica, during the past five years. Fairly full notes were taken of 121, and it was on these notes that he chiefly depended for statistics.

Strachan first describes the usual symptoms exhibited. The patient complains of "numbness," and a "burning heat" in the palms of the hands and the soles of the feet. The numbness is often accompanied by "cramps" and is worse at night and during wet weather. He complains also of impaired vision and hearing, and often of a feeling of constriction around the lower part of the chest. Usually an eczematous condition of the tops of the eyelids, the angles of the mouth, and the muco-cutaneous margins of the nostrils will be noted. The lips are unusually red, and the palms of the hands hot to the touch and hyperæmic. Should the case be one fairly advanced, there will be complained of, in addition, inability to walk well and incapacity to work owing to failure of motor power in the upper extremities. Such cases suffer most from constant pain in the extremities, especially the feet. One can almost pick out the cases of malarial peripheral neuritis by noting those patients who are sitting up in bed rubbing their feet, and moaning and crying. A patient in the more advanced stage will be carried to the hospital by friends. There is extreme wasting of the muscular system. He cannot move or even feed himself, any attempt at movement resulting in a peculiar, aimless jerk. The pigmentation of the skin is increased, and there is marked pigmentation in the palms of the hands, soles of the feet and lips, where the pigment is normally less in quantity in the colored races, than in the rest of the skin. Respiration is impaired owing to the condition of the respiratory muscles, all of which are called into play to aid in procuring the necessary amount of air. Should such a patient die, it is most often from the paralysis of the muscles of respiration becoming complete, or the heart being allowed to run riot without the restraining influence of the vagus which has become involved. Death is, however, a rare termination of this form of neuritis; recovery, more or less complete, being the rule.

Strachan next examines more in detail the various points noted in the general description given, discussing first the subjective symptoms, such as, (1) dimness of vision; (2) impaired hearing; (3) numbness and cramps in the extremities; (4) girdle pain; (5) joint pains and other abnormal symptoms or sensations. Certain objective symptoms or signs are further discussed: (1) trophic changes; (2) monoplegias; (3) altered gait; (4) conditions of patellar reflex; (5) conditions of cutaneous reflexes; (6) conditions of sensations; (7) soreness of muco-cutaneous lines of junctions; (8) wasting of muscles.

In a few cases Strachan had seen bullous eruptions on the hands and feet, and in others small ulcers which had evidently resulted from untreated bullæ. Sometimes small scattered vesicles were seen on the fingers and toes. Desquamation of the palms and soles had been noted during convalescence. In two or three cases corneal ulcerations occurred. Facial paralysis was present in seven cases; in one double facial palsy was a marked feature, The patient recovered perfectly. In one case there was paralysis of the external rectus in each eye. The gait in all but very mild cases was markedly altered from the normal. The knee-jerk was absent in more than half the cases presenting themselves (53 per cent.); was exaggerated or subnormal in 23 per cent., and was normal in the rest. The condition of the cutaneous reflex excitability varied greatly—was exaggerated, diminished, absent or normal. Pain was almost always present. Even in very bad cases the prick of a pin was felt, although there was delay in the transmission of sensation. Sensations of touch, heat and cold, although delayed, and the former blunted or impaired, were only in the most severe cases completely absent. Soreness of the muco-cutaneous borders, *i.e.*, of the eyelids and lips (commonly), urethra, anus, or vulva (rarely) was almost the first indication that the patient was attacked. Wasting and contraction of the muscles was very marked in extreme cases, the "claw" hand and foot being prominent features. The changes revealed by the ophthalmoscope were varying degrees of retinal hyperæmia, rarely amounting to optic neuritis. Strachan had not seen optic atrophy, but there was generally an increase in the pigmentation of the fundus. Pigmentation of the brain and spinal cord was the only feature noted post-mortem. Hyperæmia due to dilated peripheral vessels seemed to be an early feature, if not the earliest. The congested conjunctiva, and muco-cutaneous borders; the deep pigmentation which remained in so many cases as an evidence of the past disease, together with the pigmentation noted at the post-mortem examinations would support the idea of long-continued dilatation of the peripheral blood vessels.

Strachan concludes that the circulating poison which starts the nerve changes in this form of peripheral neuritis is the poison of *malaria*. It is the more chronic form of malarial poisoning which is followed by neuritis, just as the more chronic form leads to anæmia and renal changes, more frequently than the acute. By chronic is meant the almost daily occurrence, for varying periods of time, in persons living in malarial districts, or who have suffered from some intense form of malarial poisoning, of slight ague attacks, unattended by rigor and only followed by brief and slight sweating. Often the patient is only conscious of a feeling of lassitude and pain in the limbs. This going on for a long time, with at intervals a more acute attack, the patient finds himself growing feebler, anæmic, and suffering from ill-defined sensations of malaise. Such patients have sometimes enormously enlarged spleens.

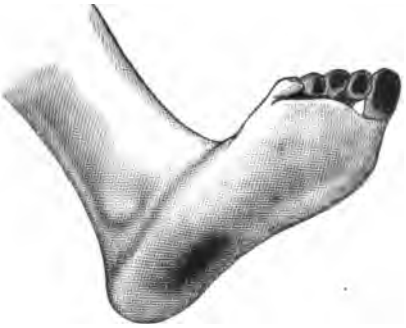
The drugs which relieve or cure are anti-malarial,—especially quinine, perchloride of mercury, and arsenic. Good food is also important.

EXPERIMENTAL NEURITIS.

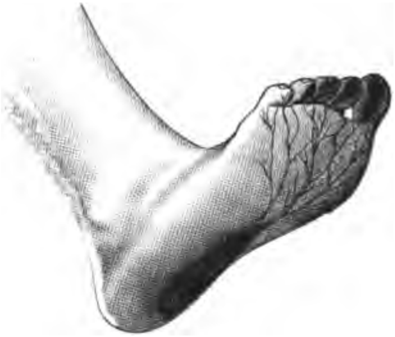
Pitres and Vaillard,[44] to whom the subject of multiple neuritis is indebted for previous valuable investigations, have made a series of interesting experiments on the effects of hypodermic injections of ether in the production of neuritis. These experiments throw new light upon the subject of traumatic neuritis, and in addition have a practical, admonitory value to those using hypodermic injections, particularly of sulphuric ether. Cutaneous anæsthesia, disorders of motility, and even serious trophic lesions, have been observed following such injections, and the experimental researches of Arnozan and Salvat proved that the mechanism of these lesions was a neuritis. When an injection of half a cubic centimetre of sulphuric ether is made deeply into the cellular tissue which separates the muscles on the back of the thigh of a guinea pig, paralysis of sensation and motion results in the parts of the limb situated below the level of the injection. Generally the anæsthesia occupies the two outer toes and the outer aspect of the leg. After a few days these phenomena may be accompanied by œdematous swelling of the foot, ulceration of the toes and tarsus, falling off of the nails, etc. The anæsthesia and the paralysis are manifested immediately after the injection, and in a short time they have reached their fullest extent, and, once developed, persist for several weeks or months. Pitres and Vaillard found, on histological examination, the nerve above the zone of lesion normal; at the level of the injection various alterations; and below Wallerian degeneration. One of the four cases reported by Kast was an example of acute neuritis after an ether injection.

PLANTAR NEURITIS.

Hughes,[45] of St. Louis, under the name of *Plantar Neuritis*, has described a rare, painful and formidable malady of the terminal distributions of the two popliteal nerves in the foot, briefly referred to in the SATELLITE for August, 1887. The history of two cases is given in detail. We give three figures taken from the article of Hughes. Fig. 1 shows the painful area in the first case; the intensest pain being at the darkest spots. There was no erythema in this case, but pallor of the foot and toes. The shaded parts in Fig. 2 shows area of flushing in the second case described, and region of pain, the chief pain being at the darkest points. Fig. 3 is a diagram of the digital nerve distributions and their trunk connections with the plantars and beyond, showing how the internal aspect of the fourth toe is supplied by branch from the plantaris internus. We must accept, according to Hughes, the term neuritis or "neuralgia plantaris" for these cases, or apply to some of them a new designation, as they are often, as in the cases here described, instances of evident constitutional neuropathia with local neuralgic pain, for both patients were hyperæsthetic to sound as well as to touch; both were mentally very irritable, and one had marked cardio-gangliopathic irritability.

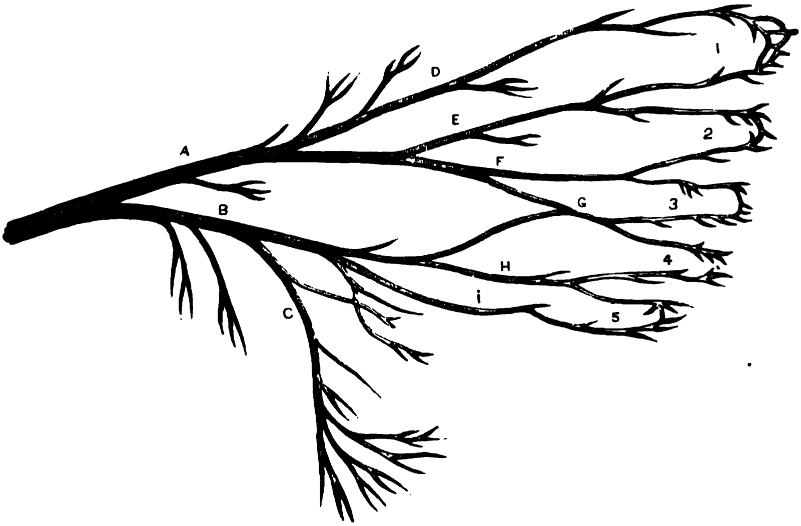


Painful Areas in Case of Plantar Neuritis.—(Hughes.)



Areas of Flushing and Pain in Case of Plantar Neuritis.—(Hughes.)

(*Western Medical Reporter.*)



The Digital Nerves and their Trunk Connections.—(Hughes.)

A. Internal plantar nerve. B. External plantar nerve. C. Deep branch of external plantar nerve. D. First digital nerve. E. Second digital nerve. F. Third digital nerve. G. Fourth digital nerve. H. Fifth digital nerve. I. External digital nerve.

1. Branches of digital nerves to first toe. 2. Branches of digital nerves to second toe. 3. Branches of digital nerves to third toe. 4. Branches of digital nerves to fourth toe. 5. Branches of digital nerves to fifth toe.

NEURALGIA.

The most valuable paper on neuralgia during 1887 is that of Dana,[46] entitled *A Clinical Study of Neuralgias, and of the Origin of Reflex or Transferred Pains.* Valuable charts and diagrams accompany the paper. As the literature of this topic for a good many years had been mainly devoted to the therapeutics, it had seemed to Dana possible that a new study of some of the clinical aspects of the disease might be of interest. In particular he had hoped to begin at least a collection of facts which might show whether neuralgia in this country and climate presented the same physiognomy that it did in Europe, whence had been got the basis of most American writings and views of this disease. He had also taken up with especial care the subject of the so-called reflex origin of neuralgia and neuralgic pains. We can only give a few of his points.

True idiopathic neuralgia was a rare disease, making not over 2 or 3 per cent. of the various forms of nervous disorder. Symptomatic neuralgias, reflex or transferred pains, and neuralgic pains from toxic causes are extremely frequent, and make up over 10 per cent. of the total of diseases for which the neurologist is consulted. The number of cases collected by Dana was 453. He found, as compared with the statistics of Classin, of Kiel, who collected 434 cases, 10 per cent. more trigeminal neuralgias, and slightly more intercostal neuralgias, while the per cent. which Classin gives of sciaticas, and of all neuralgias except trigeminal, sciatic, and intercostal, was almost exactly the same as Dana's, proving in a striking way the general representative nature of the collected cases. The distinctions between neuralgic, myalgic, and neuromyalgic pains are important, according to Dana, from a therapeutic point of view. In the purest types of intercostal neuralgia, antirheumatic remedies rarely do good, while the neurotic and anodyne drugs check it very rapidly. A study of the various pains in the back and sides leads to the therapeutical aphorism,—viz., plasters are for the back, blisters for the side. This means simply that most side pains have a predominating neuralgic element, while most back pains are myalgic. Unfortunately space will not permit of a fuller abstract of this important paper.

Neuralgic Headache with Apparitions of Unusual Character.—Dr. S. Weir Mitchell[47] in describing these cases says that the ordinary subjective images of zigzag lines and rotating wheels are replaced by more definite shapes, so as to sometimes induce the belief on the part of the patient that a ghost has been seen. In two persons instanced the vision came as the only visual prodrome of severe headaches. In another the appearances were various; at times followed the common zigzag, and at others occurred in the intervals of a series of exasperating headaches. Some of the details with reference to these cases are highly interesting.

Migraine followed by Temporary Paralysis of the Third Nerve.—Suckling[48] reports a case of migraine followed by a temporary paralysis of the third nerve. The patient, a youth of 18 years, had suffered since infancy with severe headaches, increasing in frequency. An attack lasted two days. He felt a slight pain above the left brow, which gradually increased in intensity and was followed by a flow of saliva into the mouth; he felt very cold. He had to remain in bed for two days without sleep or food. The pain attained its maximum in twenty-four hours; it then as gradually subsided. He complained of a nasty taste. After an attack the scalp was very tender. The eye affection was first noted in infancy, and had frequently recurred. The left eyelid began to drop when the pain was wearing off; it took twenty-four hours to drop completely and in another day had usually recovered. The eye had twice turned outward. Guarana in 30 grain doses, taken every hour at the commencement shortened the duration of the attacks and they had not been followed by paralysis.

Migraine in Childhood.—Dr. Wharton Sinkler,[49] of Philadelphia, holds that migraine is more common in children than is generally realized. Popularly, and by some physicians, the attacks of "sick headache" which many children have are attributed to disorder of the stomach from some indiscretion in diet. The children who suffer from migraine often belong to neurotic families. Dr. Sinkler had under his care for sick headache a lad of fourteen years whose mother had violent attacks of neuralgia, and one of his sisters was a well marked case of hysteria. In many instances ocular defects are present; these cause eye strain, and the attacks of migraine become more and more frequent in proportion as the eyes are used, until the defect is corrected by glasses. It is not in all cases, however, that headaches which follow excessive use of the eyes are due to ocular defect. Migraine from eye strain is not uncommon in children. Precocious sexual development in either sex often leads to this form of headache. The attacks are markedly paroxysmal, occurring from two to six weeks apart, and become more or less frequent, according as the conditions for their development are favorable or otherwise. If a child has already begun to have attacks of migraine, nothing is of more value than attention to general health. Such children are often pale and thin, and have but little appetite. If change of air can be secured, it is often enough to obtain relief from the attacks. If the patient cannot be sent away, tonics, good food, cod-liver oil if borne, or butter and cream are advantageous. Bromides in small doses, long-continued, are sometimes useful.

Treatment of Neuralgias and other Painful Affections.—Laura[50] reports a case of facial neuralgia, accompanied by serious flooding in a young woman, 25 years of age, of strong constitution, accustomed to an active life. She had been suffering for several days. Laura gave her Chanteaud-granules of aconitine, one granule every half hour, the doses to be given at longer intervals as the pain became easier. Acidulated beverages were given. Repose of mind and body were enjoined. In order to test the efficacy of the Chanteaud-aconitine in this case, no treatment was ordered for the flooding, which was due to simple functional disorder. The aconitine granules were taken very regularly as prescribed, and fourteen hours later, the pain was so considerably diminished that she was quite calm and passed a good night. The next morning the pain had disappeared. The menstrual flow continued, but was weaker. Chanteaud-granules of ergotine, and vaginal injections of water at 50° C., with rest, caused it soon to cease completely.

Fussell[51] reports two cases of malarial neuralgia in the first of which 36 grains of quinine were administered in the afternoon. The second day after the patient was thoroughly cinchonized, but free from pain, chill and fever. She was put on gradually decreasing doses of quinine and arsenic, as a result of which the pain never returned. In the second case the patient was put upon three grains of quinine every three hours, with increasing doses of arsenic. In a week the dose of quinine was doubled and the arsenic continued for another week. The quinine was gradually decreased and the arsenic continued, the treatment resulting in cure at the end of the month. Burr,[52] resident physician of the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases, reported for Osler a case of supra-orbital neuralgia in chlorosis cured with Blaud's iron pills.

Salzer,[53] assistant to Prof. Billroth, relates a case of a severe trigeminal neuralgia in which medicines, galvanization, repeated sections of the buccinator and the zygomatic nerves, and even the ligaturing of the left carotid artery, had no effect. Dr. Salzer relieved the patient by a resection close to the foramen ovale, of the third or inframaxillary branch of the fifth.

J. C. Wilson has published a note on *antipyrin* in the treatment of sciatica. He has recently used antipyrin in three cases with gratifying success. An abstract of this note is given in the SATELLITE for February, 1888. The *Medical and Surgical Reporter* for October 8, 1887, contains a highly interesting communication on the successful treatment of painful affections of the head (cephalalgia, migraine, tic douloureux) by antipyrin, by Prof. Germain Sée. He directed attention to the master symptom, the cerebral pain, which he endeavored to combat without attempting to reach the cause. He gave facts with reference to 42 victims of migraine, which with most of them was hereditary.

Seifert[54] has an article on antifebrin in which he says that this substance has a paralyzing effect after initial stimulation; anæsthesia and analgesia follow. He quotes Weil and others. Lépine noticed weakness in the hind legs in animals and also anæsthesia, seldom convulsions. Bokar concludes from experiments that antifebrin has no effect on the brain, but a paralyzing effect on the sensory portions of the cord. Krugn used it first successfully for neuralgia of the face. Lépine had a similar result in the lancinating pains of tabes dorsalis, after one or two doses of eight grains. Dujardin-Beaumetz reports success in irritable states of the spinal cord, in "épilepsie spinale." Secrétan, Faust, Sippel, Widowitz and Weil report success in occipital neuralgia, migraine, facial neuralgia, the restlessness of sick and feverish children, and epilepsy. Dujardin-Beaumetz recommends it in rheumatic pains in from eight to fifteen or even twenty grains. Salm has not had any result in eleven cases of epilepsy. Fisher says that larger doses ought to be used in tabes than when the drug is used as antipyretic, and that it gives relief in syphilitic occipital neuralgia, and in syphilitic dementia paralytica with racking pains in the legs; also in acute anæmic headache and in hemicrania; also in obstinate pruritus. Faust[55] extols antifebrin in migraine in doses of from one half to one gramme (8–15 grains). He could note "with his watch in his hand," how in the average of half an hour to an hour the pain ceased suddenly. He considers it much superior to sodium salicylate, antipyrin, etc. It is useless in headaches due to organic brain lesion and meningeal diseases, but good in congestive forms of the affection. It is very useful in the headache following alcoholic excess. M. Putnam-Jacobi[56] had given antifebrin to a child with pleuro-pneumonia, and with lowering of the temperature relief from pain was also produced, although the physical signs remained and typical defervescence occurred.

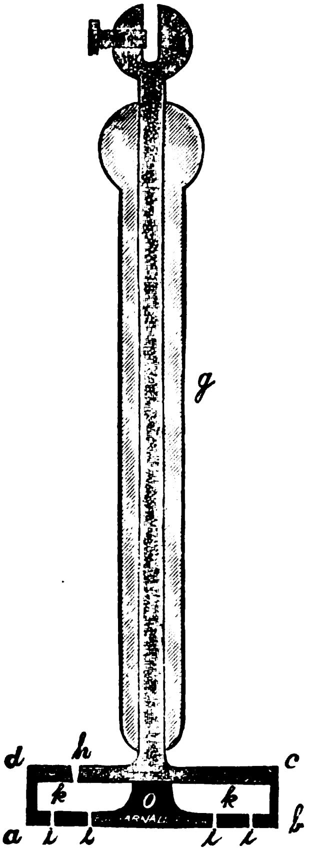
Aschenvach,[57] of Corfu, reports that suffering from sciatica in the evening, he took a dose of half a gramme of salol, and at midnight one gramme, after which he fell asleep, and remained perfectly free from his pains.

Stekoulia[58] tried osmic acid in twelve cases of idiopathic sciatica, the duration of which varied from fifteen days to two years; he had eight successes and one case much improved. An aqueous solution, containing one per cent. of the acid, is generally used, and of this about sixteen minims are injected. It stains the skin and clothes black. The injection should be made at the seat of the pain, at first daily and then less frequently. Neuher first suggested osmic acid as an anti-neuralgic, and he and Eulenberg published successful results. J. W. Brown[59] reports three cases of sciatica in which he used croton oil, to show that it is frequently dependent on simple constipation, the correction of which cures the sciatica. Rogivuc,[60] at a meeting of the Société Vaudoise de Médecine read a paper on the treatment of sciatica by chloride of methyl, in which he had good results. According to Jacquet,[61] immediate cessation of pain in sciatica can be obtained by refrigeration in the healthy leg. In ten cases he obtained six cures which lasted several days only. Debove[62] was able to afford marked relief in a case of obstinate sciatica by means of a spray of chloride of methyl applied along the course of the sciatic nerve of the unaffected member, and Raymond[63] reported a similar success. Jacoby[64] read an interesting paper on the treatment of neuralgias by intense cold (chloride of methyl, etc.) at the meeting of the American Neurological Association.

DIFFUSION ELECTRODE.

Adamkiewicz called attention to a method of treating pain occurring in the superficial nerve trunks by the application of chloroform to the affected area, over which is placed a diffusion electrode connected with a positive pole of a constant battery. Ayres[65] describes an improved diffusion electrode.

In the Adamkiewicz electrode the opening into the reservoir is very narrow and extends through an inch or more of the distal end of the handle. Besides the difficulty of getting fluids into the reservoir, it will not permit the cleansing of this chamber. In the Ayres' improved electrode this objection is overcome by unscrewing the proximal end of the reservoir at *dc*, and thus exposing the whole interior. Even porous carbon does not allow of transudation of sufficient fluid. Through the carbon plate several small apertures have been made, to facilitate the process. In the modified electrode a carbon column is made solid with the diaphragm *ab*, and abutting against the metallic plate *dc*, so that the current of electricity coming through the handle may the more readily enter and mingle with the liquid in the chamber. In the other electrode there are good reasons for believing that the current would take the lines of least resistance, viz., around *d* and *c* to *a* and *b*, and thence to the tissues.



g, handle; *h*, aperture for admission of air; *d*, *c*, metallic plate solid with handle, and screwing at *d*, *c*; *a*, *b*, carbon plate; *i*, *i*, *i*, *i*, apertures for escape of liquid; *o*, carbon column solid with *ab*.—(*Pittsburgh Medical Review.*)

DISEASES OF THE MUSCLES.

Considerable activity has been shown during the past year in the study of disease of the muscular apparatus. The reported cases, among other things, help more clearly to the differentiation of the two classes of atrophies which are now generally recognized, namely, (1) progressive muscular atrophy of myelopathic origin, which Charcot has proposed to name the Aran-Duchenne type; and (2) progressive muscular atrophy of myopathic origin which Erb, Landouzy and Dejerine have particularly investigated.

Demonstration of the Existence of Trophic Nerve Fibres.—The actual demonstration of the existence of the trophic nerve fibres, apart from vaso-motor fibres, has not until recently been made, although many evidences pointing to that conclusion have been put forward. Joseph,[66] of Berlin, in a number of experiments on cats, produces facts which go to prove the existence of trophic nerve fibres in the peripheral nerves. In order to avoid any vaso-motor changes, the second cervical nerve was selected for section, which, as will be seen, contains no vaso-motor fibres. This nerve was used in all the experiments. Mere section of the trunk of the nerve produced no effect, as reunion rapidly took place, so that it became necessary to remove a considerable portion of the trunk to prevent this occurrence. In some of the animals the ganglion on the posterior nerve-root was also removed. The changes consisted in loss of hair, at first localized, afterward gradually extending, situated above and behind the ear where the trunk was only severed; above the eye and over the cheek, where part of the trunk and ganglion on the posterior root was removed. After a time complete baldness and a shiny atrophied state of the skin made their appearance. No increased vascularity, inflammation, or any other change whatsoever could be discovered in the skin. From these experiments Joseph concluded that the trophic nerve fibres exist apart from vaso-motor fibres and entirely independent of them; and he explains the similar changes in the distribution of the fifth cranial nerve by assuming that its trophic supply comes from the posterior ganglion of the second cervical, from which fibres join the ascending root of the fifth.

Progressive Muscular Dystrophy.—In a clinical lecture at the University of Göttingen, Otto Buss[67] discusses the subject of progressive muscular dystrophy. He holds that the "juvenile form of progressive muscular atrophy" of Erb, the "hereditary muscular atrophy" of Leyden, and the "infantile form of progressive muscular atrophy" of Duchenne, are simply varieties of dystrophia muscularis progressiva. He relates the history of two cases, a brother and sister, respectively 16 and 13 years of age, whose parents were first cousins. The histories are exhaustive and show that three months' treatment resulted in no improvement. Stricker excised a small piece of the left biceps cubiti. Microscopically the muscle was pale but showed no yellowish discoloration.

Dystrophy of the Face and Head.—At the meeting of the American Neurological Association, July, 1887, Putnam,[68] of Boston, presented a case which was probably one of hyperostosis of the cranium, occurring in a young woman twenty-one years of age. Her health was apparently good, and she had no history of syphilis; her mother, however, suffered with her head. The first symptoms were severe, with intense pains in her head. Enlargement and protrusion of the eyes were first noticed about two years after the beginning of the headache. Three years ago her teeth began to fall out, and at the time Putnam saw her she had but one remaining tooth. Two years since, a purulent discharge from the ears appeared, and she could not distinguish the sound of a tuning fork. Two years after the onset of the symptoms she had a miscarriage, and one year later menstruation ceased. Œdema of the left eyelid was present and the eyes were extremely protruded, so that more than half the globe projected; there was no evidence of any paralysis of the nerves, except that the tongue moved slightly to one side. A well defined opacity was discoverable in the upper cortical lens of the left eye.

Two Cases of Primary Progressive Myopathy of the Scapulo-Humeral Type.—Two cases of primary progressive myopathy (Erb's juvenile form) of the scapulo-humeral type, recorded by Erb,[69] are peculiar in having no heredity. Case 1. Tailor, aged 28 years, thought his disease was due to hard work and exposure during boyhood. At 16 he noticed weakness in the hips and thighs, which increased and extended to the trunk and arms. Examination in July, 1885, showed marked emaciation. There was more or less atrophy of the pectorales, especially the majores, the lower part of the terites, the latissimi dorsi, serrati, rhomboidi, supra and infra spinati, common dorsal extensors, biceps brachi, and brachialis anticus. The tricipetes were less markedly atrophied and the long supinator to a high degree; the obliqui and transversi abdomini considerably; the glutei maximi, and possibly also the other glutei to some degree. The muscles of the thigh were more or less atrophied, but mostly so the quadriceps and abductor, less so the flexors, and least of all the sartorius. The ilio-psoas was weak. The muscles of the face, tongue, neck, hands, forearms (except the long supinators), leg, feet, recti abdominis and deltoids were normal. No muscles were hypertrophied. The atrophied muscles were partly relaxed and partly firm, as though crossed by connective tissue; the biceps brachii were somewhat retracted. No trophic disturbance of the skin or bones was present. The sensibility was normal in every way. The mechanical excitability of the atrophied parts was more or less diminished, being almost completely lost in the biceps brachii, pectorales majores and glutei maximi. Fibrillary twitchings did not occur. Reactions of degeneration were absent, but a diminution of the faradic and galvanic contractility was exhibited in accord with the degree of atrophy. The knee-jerks were distinct. The internal organs and the urine were normal. The patient was discharged without any change in his condition worthy of note. Case 2. A shoemaker, aged 26, in his 22d year and during his two years' military service, had weakness of the right shoulder without pain or stiffness; the pain increased and a year later the left arm became affected, the scapulæ began to project, the atrophy became noticeable and increased in severity and extent. Further details are much like those given in Case 1. The two cases were markedly similar, the only difference being that the observations of the second case were in an earlier stage than in the first one. A series of cases forming a group known as the scapulo-humeral type of progressive primary myopathy enumerated by Erb were reported by Erb, Landouzy and Dejerine, Vladimir, Roth, Alessandro Mariana, Dubois de Neufchatel, Duchenne de Boulogne, and Friedreich.

Simple Acute Muscular Atrophy.—Kast[70] (noted at some length in the SATELLITE for February, 1888) describes the rapid atrophy of the muscles after acute inflammation of the joints. Charcot considered it to be reflex, caused by functional disturbance of the spinal cord. In wounds to the quadriceps and deltoid muscles a simple atrophy was often noticed soon after. Kast found the muscles lax, and electro-excitability lowered, as well as the reflexes. No inflammatory changes or proliferation of the nuclei were observed in the muscle, but a simple diminution in size. This has been experimentally proved on animals. He thinks a vaso-motor influence has something to do with the changes.

Progressive Muscular Atrophy of the Aran-Duchenne Type.—Audry[71] contributes an interesting paper on progressive muscular atrophy of the Aran-Duchenne type, accompanied by rare symptoms, and terminating rapidly by the suppression of the respiratory muscles. He draws attention to the following points, which were particularly interesting in the history of this case: (1) The singularly rapid progress of the affection; (2) the symptoms which followed the paralysis of the respiratory muscles; (3) the subcutaneous œdema; (4) intermittent albuminuria.

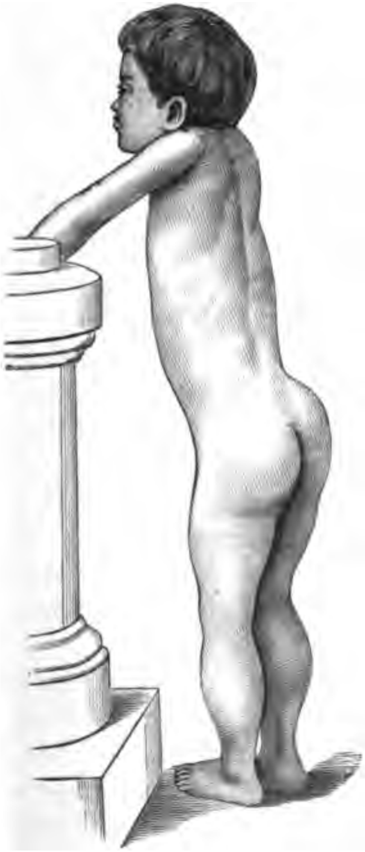
Atrophy of the Thoracic Muscles among Tubercular Patients.—Bumpar[72] has made an elaborate study of this subject based on several careful and painstaking observations. The conclusions arrived at by him are as follows: (1) Muscular atrophy is frequent among patients with tuberculosis; (2) it is found in the vicinity of pulmonary lesions and particularly at the summit of the thorax; (3) it presents certain special characters; (4) it may be phenomenally precocious; (5) it is sometimes accompanied with severe pains in the muscles in the location of the atrophy; (6) it constitutes a complication of pulmonary phthisis, in that it increases the difficulty of respiration, and perhaps contributes to hasten its evolution.

Tabes Dorsalis and Muscular Atrophy of One Upper Extremity.—Remak[73] says that atrophy of the upper extremities usually occurs only after tabes dorsalis has existed in the lower limbs for some time. The first symptoms are in the ulnar region, then other lesions of sensibility occur, so that the patient cannot tell about the movements of his fingers. In these cases both the columns of Goll and Burdach are involved. The question arises why there is no muscular atrophy in many of these cases, in spite of the extensive changes which have occurred in the nerve trunks. In cases of tabes with atrophy of the upper extremities, the ataxy of these limbs has not been most marked. Remak related the following case: A cigarmaker, aged 39, who denied syphilis, and had been a moderate drinker, had lancinating pains, gastric crises, unsteady gait in the dark or with his eyes shut, ataxy in the legs and the first three fingers of the right hand, disturbed sensibility, retarded sensation to pain, inflexibility of the pupils. The arms showed no ataxy, but there was atrophy of the abductor and opponens pollicis, and the muscles of the thenar group supplied by the median nerve, as well as anæsthesia of parts supplied by this nerve. This case showed a neuritis of the median nerve as a complication of the tabes dorsalis. This seems to show that certain motor disturbances in the early stage of tabes do not depend upon centric but upon peripheral causes. As this patient was a cigar-maker, finishing from 500 to 600 cigars a day, whereby the thumb and forefinger were in constant movement, it becomes a question whether the complication was not due to the constant over-exertion of one group of muscles.

A Rare Case of Chronic Anterior Poliomyelitis in the Adult passing into Bulbar-Myelitis.—Buss[74] reports the case of a woman aged 32, who was taken with paresis first of the right then of the left leg, followed by weakness of the right arm; the right leg then became perfectly paralyzed, with very slight affection of sensation. Ultimately both legs were lamed, with characteristic electric responses. The sphincters were not involved. The abdominal muscles, then both arms, then the interossei became involved. Later came symptoms of bulbar paralysis, as impairment of the respiration. Buss excluded multiple neuritis, because of absence of sensory symptoms and absence of pain on pressure upon the nerve trunks, and especially because of the electric responses.

Pseudo-Muscular Hypertrophy.—Hashimoto,[75] of Tokio, Japan, previous to October, 1885, had never seen pseudo-muscular hypertrophy in Japan, but at this time a girl came to him for treatment who had the pathognomonic signs of this affection so markedly that he was able to make a diagnosis on sight. He goes into a discussion of the entire subject. Griesinger's work appeared in 1865, though some mention had been made of the disease previous to that time. According to him, Billroth was the first to institute microscopical examinations of the changed calf muscles. Griesinger also examined them microscopically and macroscopically. They showed that although there was an apparent hypertrophy, strength was actually decreased. Since then several papers on the subject have appeared. Charcot and Cohnheim have made autopsies in the cases and published the results, according to which, however, it is impossible to positively locate the disease in the spinal cord or muscles as no lesion of the cord has been observed. Gowers and Lockhart Clarke have lately determined that a change takes place in the lateral gray substance of the cord, and especially in the lower part. As the author had no post-mortem experience in these cases, he felt unable to say anything about their etiology. He excised a piece of the calf muscle, under strict antisepsis, resulting in closure of the wound in two weeks.

Two theories prevail concerning the change in the muscles in this disease. According to one, the nuclei of the internal perimysium and of the connective tissue proliferate and thus cause atrophy of the muscle fibre by pressure; while according to the other, the muscle fibres atrophy and undergo fatty degeneration independently of the proliferation of the perimysium and connective tissue nuclei. Hashimoto's preparations demonstrated the complete proliferation of the adipose tissue and nuclei. They also showed a marked thickening of the vessel walls and an increase in their nuclei. He supposed that the proliferation of the adipose tissue was the result of the nutritive disturbances due to the lessened calibre of those vessels that have their walls thickened.



Pseudo-Muscular Hypertrophy (Hashimoto).—(*Zeitsch. f. Klin. Med.*)

A girl of 7 years of age had a badly developed nervous system, besides very defective speech. Her temperature was one degree lower than that of a healthy girl. She never could walk. He therefore concluded that she became affected shortly after birth. When four years of age she had a fever for four days, during which time she ate nothing and at the end of which she slept twenty-four hours. She could not advance her body either upon her belly or legs. Her mind was imperfectly developed. The calf muscles, adductors, and glutei were strikingly large, while the breast and back muscles were wasted and the upper arm muscles had knotty thickenings; the skin of the lower extremities was sensitive to cold and heat, but the muscles had no electrical reaction.

Dupuytren's Disease coexisting with Paretic Dementia.—Régis,[76] of Bordeaux, read a note at the last meeting of the Association Française pour l'Avancement des Sciences, on a case of

Dupuytren's malady coexisting in progressive general paralysis, which he summarizes as follows: (1) Dupuytren's malady (retraction of the palmar aponeurosis) is really, as there is a tendency to believe, the manifestation of a general state which is nearly always that of arthritism, and not a local affection of traumatic origin. (2) The manifestations of gout and those of general paralysis may coexist and be confounded in the same subject, so that one is forced to conclude as to the reality of a morbid relationship already admitted to a certain extent, and which new facts will certainly confirm and more and more elucidate. It was observed that diabetes frequently accompanied the retraction of the palmar aponeurosis.

Treatment of Retraction of the Palmar Aponeurosis—Dupuytren's Contracture.—Kocher[77] has an elaborate article on this subject. He says that it is very important for treatment to know which tissue is diseased. Baum says that it is the skin. Kocher the palmar aponeurosis. He reports 4 cases in which operation has been successful and in which it was proved that the aponeurosis was the affected tissue. The skin was divided longitudinally and separated from the diseased palmar fascia, and thick, hard, projecting knots and cords. The palmar fascia and its offshoots were cut out as far as they were changed and influenced the flexed position of the fingers. The cases are given in detail. He advises against postponement of the operation lest ankylosis of the joints occur, and after the operation directs the fingers to be maintained in the extended position.

Unilateral Atrophy of the Hypoglossal Nucleus, with Demonstration.—Westphal[78] demonstrated an interesting specimen from a patient who had had ophthalmoplegia externa and complete paralysis of both eyeballs. He had also bulbous appearances, and an atrophy of the antero-lateral portion of the left side of the tongue. The specimen showed the right hypoglossal muscle to be well developed, while the others were only fragments, and very small collections of the large multipular ganglion cells. On the right side the root was distinctly seen running to the nucleus, but nothing could be seen on the left, except with a very high power, and then only a few nerve fibres. He also found a gray degeneration throughout the whole length of the cord.

MYOSITIS.

On Idiopathic, Acute, Purulent Myositis.—Walther[79] refers to the fact, well known since Virchow's investigation in 1847, that the muscles may be the seat of acute inflammation. If the contractile substance undergoes retrograde changes, then the process in the connective tissue is more active, as the exudate is formed here which becomes changed either into fatty tissue, cicatricial tissue or pus. The disease may be circumscribed or diffused, chronic or acute. The causes may be pyæmia, trauma, and others which cannot be described, and which are considered to originate the so-called idiopathic forms. It is related to acute, purulent periostitis, acute osteomyelitis, erysipelas, etc. They have in common acute inflammation tending to the formation of pus. The bacillus of Rosenbach is considered to be a cause. Overaction of the muscles, trauma and lowered strength favor the occurrence of the bacilli. All muscles of the body, as well as single muscles, can be invaded by this disease. It is a grave affection, and where many muscles are involved, requires a cautious prognosis. Where cure occurs the muscles regain their function. Erysipelas and phthisis complicated some cases. Paul Hepp[80] has an article on a similar subject.

Primitive Infectious Myositis.—Brunon[81] contributes a study of *infectious primitive myositis* in which he says that a great number of infectious maladies may present myositis as a complication. The myositis which follows typhoid fever is a type. Besides this myositis, which is called secondary, there is a primitive myositis having peculiar and distinct characters and evolutions, and which, by reason of its character, may be considered infectious. It is sometimes mixed and exists as a result of either primitive or infectious and osteomyelitis, diffuse inflammation, and infectious pseudo-rheumatism. Primitive infectious myositis may take three clinical forms; *a*, the malignant form, in which death ensues in a few days; *b*, the severe form, in which cure is possible in spite of the gravity of the general symptoms; *c*, the subdued form, which has but a feeble reaction. Weakness and overwork are the predisposing causes; muscular effort is an occasional cause; the first, however, is a general and primitive affection. The treatment ought to be prophylactic, avoiding, by means of hygiene, all causes of infection.

Other interesting papers on myositis have appeared during the year: on myositis ossificans by Helferich[82] and by Sympson;[83] on polymyositis by Küssmaul and Maier,[84] Unverricht,[85] and Jackson;[86] on syphilitic myositis, by Neumann.[87]

TETANUS.

Etiology.—The chief interest which attaches to the subject of tetanus at present concerns its etiology. The infectious nature of the disease had attracted attention long before the present epoch of microbian pathology had subjected it to the test of experiment and scientific induction, the attention of Ambrose Paré having been called to it. During the Seven Years' War vast epidemics of the disease swept over the armies. Larry (according to Audry) observed something similar during Napoleon's campaign in Egypt. In a large European city a maternity hospital, which had been badly afflicted with *trismus neonatorum*, was relieved by careful antisepsis. French veterinarians observed a marked decrease of tetanus in horses after castration and other operations as soon as antiseptic precautions were introduced. Within a very recent period Rosenbach has claimed to have discovered the specific microbe of tetanus; and Carl and Rattone have produced tetanus in rabbits by injections from the pustules of acne in a man dead of the disease two hours before.[88]

Bonome,[89] at the meeting of the Italian Medical Association at Pavia, in September, in speaking of the etiology of tetanus, said that he found the pus which escaped from the wound during an attack and which by inoculation reproduced the disease in animals, to contain species of micro-organisms,—a bacillus very minute and transparent. He applied successively all the methods known to isolate one of these bacilli, but it was almost impossible to obtain one entirely pure, and he had to content himself with some in a state of putrefaction. The inoculation with these produced the true tetanus, but when he used the bacillus in a state of putrefaction alone it did not produce the disease: he believed that he had found the true specific pathology of tetanus in this organism. M. Giordani obtained the same results as M. Bonome. He persevered in his experiments of cultivating the bacillus and finally obtained an isolated one.

Verneuil[90] declared that he was convinced of the non-existence of spontaneous tetanus. In fact, a trauma quasi-microscopic—the prick of a sewing needle, hypodermic syringe, thorn, scratch or excoriation, measuring only a few millimetres—is easily followed by a true traumatic tetanus. Various pathological lesions, as burns, frost-bites, simple or specific ulcers, either primary or consecutive to an inflammatory or ulcerous abrasion of the skin, with the concurrence of a determining cause, may become the starting point of tetanus, which may be called pathological, but not spontaneous. Wounds and lesions may generate tetanus even after complete cicatrization more or less ancient. Verneuil is of the opinion that tetanus is related to virulent or infectious microbian diseases. He believes there is always a specific cause, a virus coming from without.

Shakespeare,[91] at the last International Medical Congress, gave the details of his inoculation of rabbits with material from the medulla and spinal cord of a horse which had died of traumatic tetanus. The injections were made into the cerebral dura-mater, subcutaneous tissue and muscles. His researches tend to show with other experimenters and observers, that traumatic tetanus in the horse and mule is at least sometimes an infectious disease, transmissible to other animals. The virus is contained in the medulla and spinal marrow. Dr. Shakespeare did not commit himself to any conclusions as to the prophylactic effect of the inoculations.

Page[92] has some notes on idiopathic tetanus,—the case of a young man whom he had under his care. He regarded the disease as a nervous one, depending upon an exaltation of the polarity of some portions of the gray matter of the spinal cord. Making use of the facts of Shakespeare's experiments, he believed that the specific poison of traumatic tetanus was stored in the medulla oblongata, and suggested that a similar line of experiments should be carried out in the comparative anatomy and pathology department about to be established in the new College of Medicine in Newcastle.

Peiper[91] refers to Beumer's experiments on mice and rabbits in producing trismus by transplanting material from the navel-wound of an infant dead of the disease. He says that trismus is an infectious disease just like traumatic tetanus. Peiper reports a case of which the following is a very brief abstract. An infant developed tetanic symptoms five days after birth and one and a half days after the falling of the cord. The following day the face, mouth and limb muscles were involved. The finger could not be inserted into the child's mouth. The navel wound did not exhibit anything unusual; it was covered with a slight suppuration. Death ensued about the end of the second day. The navel was excised and six mice were inoculated. Infectious tetanus appeared in three mice in 20½ hours after the inoculation. The animals were rigid in almost all their muscles. Death quickly followed. Guinea pigs were inoculated from the dead mice and in five days they died. He concludes that prophylaxis imperatively demands a careful antiseptic treatment of the navel-wound.

Analogies of Tetanus with Sunstroke.—Hiller[94] writes that tetanus has several analogies with sunstroke. Both are characterized by increased heat production and insufficient heat abstraction. The highest bodily temperatures are peculiar to both. Both are almost alike as regards mortality. In both the cause of excessive temperature is due to increased muscular contraction, and the insufficient heat abstraction. It is fair to assume that the danger in both is due to high temperature. Fever patients with a temperature of 41° C. are always in danger, and as in these two affections a continuous temperature of 41° C. for some time is not uncommon, the excessive temperature readily explains their great mortality. The therapeutic indication is to abstract heat. Hiller does not recommend cold baths for this purpose, because the cold would unduly excite an already irritated central nervous system, but lukewarm baths (at 20° R.) combined with the antipyrites. Of the antipyretics he prefers antipyrin and thallin, because they can be given for days in efficient doses without detrimental effect to the organism. Pilocarpin could also be advantageously utilized by subcutaneous injection to induce diaphoresis.

On the Behavior of the Sensory Nerves in Tetanus.—The action of the sensory nerves in tetanus, according to Erb and Hoffmann,[95] has received no consideration up to the present time; yet many symptoms indicate unequivocally that the disturbances of sensation are due to pathological processes going on in the sensory nerves at the onset as well as during the spasm. The paræsthesia, creeping sensations, tearing pain, etc., all indicate this. Hoffmann discovered that the electrical excitability of the nerves in tetanus was due to a relatively very rapid metamorphosis. That the electrical excitability of the motor nerves is increased in this disease, is directly evident from the high minimum excitability. Upon *a priori* grounds, the same would probably be true of the sensory nerves, though evidence of this effect was lacking. Only Erb held that the law of sensory twitchings corresponds strikingly with the motor. Comparison of Erb's results, mentioned in his *Electrotherapie*, from his experiments on the superficial radial nerves, and Hoffmann's upon tetanus cases, resulted in the conclusion that the galvanic excitability was increased. Central examination of healthy persons compared with the electrical reactions from a tetanic case, convinced Hoffmann of the correctness of Erb's statement, and taught that the *electrical excitability of sensory nerves in tetanus is increased* in the same way as is known to be the case with motor nerves since Erb's experiments. Hoffmann had two later cases of tetanus which justified his former conclusions.

Treatment of Tetanus.—Marestang[96] reports at considerable length a case of "chronic tetanus of a light character" in which he derived good effects from hypnotism. The patient was a young man aged 24. The reporter discards the idea of the trouble being hysterical because especially of the absolute integrity of intelligence and sensibility, and the absence of neurotic antecedents.

Lopez[97] reports the case of a man with marked opisthotonos and painful cramps, for whom chloral and morphia had been prescribed in vain. Finally three syringefuls of the mixed solutions of morphine and cocaine (each 5 per cent.) were injected. The effect was immediate. After two hours he could move the limbs, turn in bed and open his month. The patient rapidly recovered, and in one week's time returned to work.

Brunäuer[98] reports the case of a woman with rheumatismal tetanus cured by pilocarpine. He practiced injections of this drug in doses of 2 centigrammes a day. Violent perspiration was induced and prolonged for six or eight days. During the night hydrate of chloral was administered. At the end of nine days' treatment the convulsions had completely disappeared, and the patient was able to open her mouth.

CHOREA.

The most conspicuous and comprehensive paper on this subject in current medical literature is the Report of the Collective

Investigation Committee of the British Medical Association,[99] which has been prepared by Dr. Stephen Mackenzie. The results are based upon 439 cases. There can be no doubt that the collection of facts in this inquiry is of great value, yet it appears not to settle definitely any of the numerous points on which opinions are divided with regard to the causation, pathology and phenomena of chorea. It is hoped that it will serve as a basis for future inquiries, and narrow the issues for future investigations. Such are the collator's hopes and words in his opening paragraph.

It is obvious that some of these inquiries embody much more useful questions than others, and we will devote space briefly to the most useful alone.

Etiology.—The preponderance of females over males—nearly 3 to 1—is very marked, while the age of incidence is decidedly from 6 to 15 years, the percentage of cases occurring in this period being 77.46. The disease occurred at exceptional ages at 32, 40, 63, 68, 73, 78 and 86 years. The "lower classes" of society—which is probably English for the laboring classes—furnish 70.46 per cent. of these cases, but as that is probably about the percentage of these "classes" to the total population these statistics have not much significance. The moderately strong and the fair-complexioned suffer most, as do also the average good intellects, and those whose growth is moderate. Disorder of the sexual function in the female appears to be a causative element, and may partly account for the preponderance of the disease in that sex, but the numbers are too small to warrant a conclusion. The cases of chorea in connection with pregnancy are of great interest, and are related in detail: 5 recovered, 1 died and 1 was lost sight of. Several aborted or went into premature labor. The movements in some were very violent. The fatal case had premature labor induced by Barnes bags; the choreic movements, however, did not cease.

Antecedent illnesses form one of the most important sections of the inquiry. Rheumatism with distinct joint affection and fever had been present in 26 per cent., and about half that number had had vague rheumatic symptoms. Scarlet fever had been present in 29 per cent., measles in almost as many, and anæmia in 20 per cent. Many other diseases had been observed in these 439 cases, but they had no especial significance. Many of the rheumatic cases occupied debatable ground. It is, however, of special interest to know that a certain small number of cases were reported in which rheumatism appeared first during the choreic attack in patients who had never before had rheumatic symptoms. In the rheumatic cases 50 per cent. had organic heart disease, and functional heart disorder occurred in 14 per cent. These, as would be expected, gave a much larger percentage of heart disease than the non-rheumatic cases—50 per cent. in the former to 32 per cent. in the latter. Scarlet fever had been present in 29 per cent. Considerable interest, says Dr. Mackenzie, attaches to the influence of the exanthemata in the causation of endocarditis, which in turn might cause a murmur before and independent of the chorea, or predispose the heart to a production of a murmur under the choreic influence. To be brief, the testimony of the statistics is to show that scarlet fever predisposes to the occurrence of heart affection in the attack of chorea. There is no evidence that measles *per se* induces heart disease in chorea. For exciting causes we have 98 of fright, 71 of mental overwork (at school), 17 shock, 13 of imitation, and numerous others, making a total of more than 50 per cent. neurotic causes. Among a variety of other causes given many were probably only coincidents, and the bulk had small value. One case was ascribed to the east winds.

Varieties, Duration and Course.—Thirty cases were hemichoreic (quite or almost), right-sided in 10 cases, left-sided in 13, and not specified in 7. In 2 cases the movements were entirely facial. Details are given of some cases of special interest. The usual duration of an attack is shown to be from two to three months. Death occurred in 2 per cent. of the cases, and of these 9 fatal cases one case was 78 years old, another was the puerperal case already alluded to, and in several others there was heart involvement, embolus, or lung complication.

Complications.—The heart symptoms of these cases are worked out in elaborate tables, some of which are not very clear or instructive, especially the one which relates to the existence of heart diseases before, during and after the attack. Mitral disease is the most common form. It appears from one of these tables that the heart can be permanently (or for a long time) impaired by an attack of chorea previous to which it had been normal. It is of special interest to know that at least 12 per cent. of these cases were associated with rheumatism *during* the attack. In seven of the whole number of reported cases, "rheumatic subcutaneous nodules" occurred in connection with the attack of chorea. A long list of other complications is given, ranging through the whole nosological list, headache being first with 12 cases. As the others were never present in more than one or a very few instances it is proper to judge them mere coincidents; such, for example, as toothache, constipation, spinal caries, dyspepsia and lupus. It would, however, have been of great interest to know more about the mental condition of these 439 cases, during the attack and it is to be regretted that this was not made the object of special inquiry. This list of complications contains the only light on this question as follows: hysteria 3; emotional disturbance 2; ungovernable temper 1; sleeplessness 4; delirium 1. But there certainly has been observed in chorea more decided mental impairment than is included in this meagre list. Four cases are given in detail in which speech and articulation were affected,—a surprisingly small number, and suggesting a lapsus on the part of the observers. Two of these cases had aphasia. One case of paralysis of the arm is given, but not much light is thrown upon paresis following chorea. Two cases of epilepsy are recorded. Three cases only had worms. Urticaria occurred in 4 cases. Among common ailments to which these patients were liable, *independent* of the choreic attack, nervous effections of various kinds are especially indicated in 26 per cent., and the most common of these was headache in 18 per cent.

Family History.—The family history was examined with the following results, stated briefly: Nervous affections had existed in the patients' families in 202 cases, of which 143 were in a direct and lineal descent. One remarkable family history of chorea is given, affecting several generations, but it may be a question if the inveterate hereditary shaking recorded was true chorea. It may also be a question whether the cases of this disease confined to the face were not instances of the so-called habit chorea.

Treatment.—There is no specific for this disease. Arsenic and iron are most used, partly, it appears, because they are the fashion. Both of them fail in some cases. Treatment without drugs and with full diet alone gave an average duration of ten weeks, which is about the average of treatment with drugs. Arsenic is recorded to have done good in only about three per cent. of the cases,—which is unexpected. Salicin and salicyl compounds were recorded as of marked benefit in a few cases; but per contra it was abandoned for arsenic with great advantage in one case. Almost all cases relieved by salicin were rheumatic. Some of the other remedies were the sulphate and oxide of zinc, belladonna, chloral, gymnastics, galvanism and change of air. Cimicifuga, which is highly lauded by some American practitioners, is not as much as mentioned.

Sequelæ.—Chief among the sequelæ were anæmia, certain nervous irritable states, stupidity and diminished intelligence. This report is of interest and value, and reflects credit on Dr. Mackenzie; at the same time it shares the weakness and errors inseparable from the statistical method. It has been said that one man with an idea is better than a million without it, and so in statistics. One case positively demonstrating a fact is better than a million, all equally obscure on that fact. The subject of the exact pathological relationship of chorea, rheumatism, heart disease and scarlatina is not much advanced, although perhaps it is emphasized in this report; while the casual relationship of fright and certain nervous states to chorea is indicated but not proved. The conclusions as to treatment are commonplace and without novelty, excepting as they indicate a certain value in salicin, and confirm the use of a few old and favorite drugs.

The Seasonal Relations of Chorea and Rheumatism.—Lewis[100] has prepared statistics on this subject which supplement most opportunely the collective report of the British Association as collated by Mackenzie. The seasonal relations were apparently entirely ignored by the Committee of the British Association in their cards of inquiry. Lewis shows that the lowest averages of onset occur in October and November, about 4 per cent.; it then rises rapidly until January, which reaches 8.2 per cent., falls slightly in February and again rises, reaching its highest point in March,—15.3 per cent. The next highest months are May and July, which each give about 10.5 per cent., and then the tracing falls steadily until October,—the lowest. In comparing the tracings of mean relative humidity, of mean barometer, or mean daily range of thermometer with the chorea tracing, not much resemblance is seen, but a resemblance begins to be apparent between the chorea tracing and the tracing of cloudy days. The tracing for storm centres represents the number of storm centres passing within a circle of 400 miles radius described around Philadelphia as a centre. This tracing corresponds very closely to the tracing for chorea given above; it reaches its highest curve in March and its lowest in August. When acute articular rheumatism (467 cases) is traced, the following results are obtained:—November has the fewest, about 5 per cent., the tracing then rising rapidly to January,—10.7 per cent.; falling slightly in February and March, to rise to its greatest height in April,—17.3 per cent.; and then gradually, with some slight irregularity, falls to its lowest point in November. While the chorea keeps pace month by month with the storm centres, rheumatism is exactly one month later, which is not what would be supposed if rheumatism is a causative element in chorea. The seasons gave the following returns: spring 152, summer 120, autumn 64, winter 101. Dr. Lewis thinks that this study may be considered as forging one more link in the obscure chain which binds together rheumatism and chorea.

In a valuable paper on the *Etiology and Symptoms of Chorea*, Osler found that 15 per cent. of the cases showed antecedent rheumatism; but in only 7.10 per cent. was the rheumatism immediately associated with the chorea.

Relations of Chorea and Rheumatism.—The relation of chorea and rheumatism is the subject of observation by Sturges.[101] His conclusions, based on 177 cases, are as follow: The intimate association of polyarthritis and chorea justifies the statement that the two affections are different manifestations of the same morbid condition. But this is only true in very early life, because this relation of the two affections is hardly discernible in adult life. Growth weakens this connection, and age breaks it down altogether. The time of life when rheumatism is most distinct and characteristic is the time when chorea is hardly ever seen. The one waxes as the other wanes. The full development of acute rheumatic polyarthritis as a widespread inflammation involving many joints at a time, is the signal that the time has come for the decline of chorea. What hypothesis is to be set up? To what law of nature must we appeal to explain this age variation? The facts must be kept clear of theories at present. What has been said claims to be based on clinical observation, which must be carefully separated from speculative comment.

Diagnosis and Varieties of Chorea.—Guinon[102] has written upon the diagnosis of chorea and establishes three grand divisions: (1) chorea major (hysterical); (2) chorea minor,—the chorea of Sydenham; (3) the group of choreiform movements, so called. The grand chorea is rare in perfectly typical cases: it would be difficult to confound it with any other convulsive affection. It presents itself in distinct attacks, which are provoked by certain special influences, such as the emotions, etc. Between the attacks the patients are in a normal condition; in the attack itself there is no loss of consciousness. The attacks are regular in their occurrence, and rhythmical; the same for each patient, whence the name *rhythmical chorea.* The movements cannot be confounded with any other involuntary movements: they are the production of acquired gestures. Varieties of this form of chorea are the dancing chorea; the *malleatory* chorea (named from the imitation of a blacksmith beating upon his anvil) and other acquired and imitative movements. The attacks are thus of a character, special to each case, and according to a mode established in advance. Other forms are described which begin rather as hysterical affections with loss of consciousness, gradually assuming a rhythmical character, passing on to acquired movements, and becoming choreic with preservation of the consciousness.

The chorea minor, or Sydenham's chorea, displays the well-known movements described in all the books. Such of its characteristics as are of great importance in diagnosis are briefly indicated. Although the movements are sudden, instantaneous in their appearance, and unexpected, there is a certain gentleness about them. They never reproduce, or imitate, except by chance, any movement of ordinary life, any automatic or acquired gesture. They are absolutely incoördinate and interfere constantly with voluntary movements, and the will cannot control them. Voluntary movements are interrupted and frustrated by these choreic convulsions. Finally these convulsions of chorea are not produced in paroxysms. Guinon lays stress upon the distinctions which he draws between the various phenomena at different stages and intensities of the disease; thus in very benign cases, or at the debut or decline of chorea, the shocks in the limbs are not present, or if present are confined to slight disturbance of the muscles of the arms and particularly of the fingers, sometimes the muscles of the face being alone affected.

Another class of the choreas exists, which still bears the name, but has no relation with the chorea of Sydenham, showing itself in children, adolescents, sometimes in pregnant women, and rarely in adults. This class exists in old persons, and is almost identical with true chorea as concerns the movements; but it is chronic and completely incurable, hence presenting a grave character not known in children. It is the expression of an organic lesion, although this lesion is not identical in all cases. Hence among choreiform diseases, he distinguishes those which are caused by an organic lesion, and those which result from a mere functional disorder of the nervous system. Among the former he includes hemichorea, which is "generally symptomatic of a lesion of the brain with degeneration of the lateral tract,"—evidently the disease which we call post-hemiplegic chorea. Hence there will often be found exaggerated reflexes and contracture. He says that athetosis can be classed with this symptomatic hemichorea. It comes from the same cause, and the one can succeed the other. It consists in incessant movements of opening and shutting the hand, finger by finger, in such a way that the patient cannot seize or hold on to an object.

Certain organic affections of either the centres or the nerves, causing movements which are localized in the domain of a motor nerve, as the facial or in the neck, he desires to see given another name than tic, as they are not true convulsive tics, and are to be distinguished as localized in a single muscle or group of muscles or the territory of a nerve. This variety may be caused by a tumor or other organic lesion.

Again, two affections are known as the *Disease of Dubini* and the *Disease of Bergeron*. The former is characterized by involuntary shocks accompanied by convulsive attacks, leaving after them a certain degree of paralysis of the members and deviation of the face, as in a hemiplegia. It is fatal in 90 per cent. of cases. Bergeron's disease is characterized by its sudden onset, the rhythmical character of the shocks, which attack especially the limbs, but which can extend to the face. There are two important characters: the first, that compression of the facial nerve suppresses the spasm of the face; the second, that the disease is benign and amenable to treatment.

The *Tic de Salaam* occurs usually in young subjects, and consists in alternate movements of flexion and extension of the head upon the neck, accompanied sometimes by rotatory movements, of a frequency equal to one hundred in the minute. The attack is announced by a true aura, and involuntary micturition occurs. It is a kind of petit mal, and may run into confirmed epilepsy, with loss of consciousness.

Guinon then refers to *Paramyoclonus Multiplex* as a "species of choreiform movement," and says it is so rare that up to that time the cases of Marie, Friedreich and Lowenfeld were the only true ones that had appeared. It consists especially of shocks of individual muscles analogous to those which are produced by an electrical discharge. As we refer to this disease in another part of this report, it is not necessary to quote more here.

The convulsive tics form an important group of choreiform movements and are briefly characterized as follow:—They are caused by the hereditary neurosis and degeneration. They affect chiefly the face, but sometimes also the limbs. They are sometimes very complex, as jumping, running and falling upon the knees. They are never incoördinate, and do not interfere with the voluntary movements. They are automatic and reflex, or reproduce actions of ordinary life more or less complicated. The patients scratch themselves, pull the beard, stroke the hair, half close the eyelids, raise the shoulders, and strike the feet upon the ground. The movements come in paroxysms and follow in the same order; they are increased by the emotions. They can be arrested by a great effort of will, but not for a long time, and only with a feeling in the patient of insupportable anxiety. To these movements may be added such phenomena as involuntary exclamations, such as *caprolalia* (the utterance involuntarily of obscene and vulgar words); *echolalia* (the utterance of language spoken before the patient); *echokinesia* (involuntary reproduction of movements). Finally reference is made to the *fixed ideas* such as *agarophobia*, *claustrophobia*, *folie du doute*, etc.; but to include these last among choreiform movements appears to us to be stretching the limits of this class beyond all reason. Involuntary hysterical movements simulate the convulsive tics and the only definite means of distinguishing them in some cases is to follow their course. The convulsive tic is incurable, while the hysterical affections are eminently curable.

School-made Chorea.—Sturges[103] gives two series of cases of chorea,—a total of 42, of which 14 were of school origin. The causes do not appear to have been altogether mental overwork, but to have been partly due to worriment and fear following corporal punishment. These are mostly children of the London poor, with a "London aspect." One is given as a type, who was overworked physically at home, with insufficient food and sleep. It is difficult to see how her chorea can be called "school-made" entirely; but some cases were literally so in the sense that they were flogged into the disease according to the barbaric methods which seem to prevail in these schools. The causes are classified as follow: (1) overschooling; (2) excitement in examination; (3) home lessons; (4) caning and other punishment. Sturges thinks the fault is partly in a too rigorous and uniform rule which does not allow for individual temperament and weaknesses, and for special circumstances.

Chorea and Psychoses.—Schuchardt[104] claims, in common with Ziemssen, that chorea magna ought to be separated from the class of chorea proper and be considered as a psychosis or hysteria. Of the connection of chorea minor and mental symptoms he gives some illustrative cases: in one the choreic and mental symptoms were the combined product of lead poisoning; in others the choreic movements were the symptoms of progressive paralysis; in others the following symptoms were present: (1) idiopathic chorea followed by a change of character, mania and hallucinations, terminating in dementia; (2) chorea and mental symptoms appearing at the same time; (3) development of mental symptoms independent of chorea, with irritability and impulsive outbreaks. This was in a boy who had left-sided chorea following a fright.

Post Hemiplegic Chorea.—West,[105] speaking of a case of post-hemiplegic chorea, described the character of the movements, their violence, the shortening of the leg, and the long intervals between the hemiplegia and the hemichorea. The movements had somewhat the character of athetosis. As a result of fright by a dog, the movements involved the face and increased in violence.

Paralysis following Chorea.—Railton[106] reports a case which he thinks is a rare sequel of chorea. The patient, aged ten, had been suffering from the disease for about six weeks, affecting chiefly the right side, though the left was not entirely free. There was nothing unusual about the attack, which yielded to four weeks' course of arsenic. When the child left the hospital she was completely well. There was no cardiac murmur nor symptoms of paralysis. She never had had rheumatism. About two weeks after leaving the hospital she began to complain of pain in her legs and feet, which increased toward evening. She suffered from "pins-and-needles" sensations at the bottom of her feet. She was easily fatigued and her walking became uncertain. Two months after, when she was brought back to the hospital, the lower extremities were found wasted and the child presented general emaciation. She walked not unlike an ataxic, doubtless from paresis of the muscles. There was dropped ankle. The knee-jerk was completely absent. Under the administration of iodides of potassium and iron and the application of galvanic electricity, the child gradually improved. As regards the nature of the case, Railton thinks it was one of multiple neuritis due in some way to the preceding chorea, an opinion which was shared by Dr. James Ross, who saw the patient at a meeting of the Manchester Medical Society. The paralysis was probably due to the arsenic which was administered for the cure of the chorea.

Monobrachial Chorea.—Wood[107] exhibited a case of monobrachial chorea, not post hemiplegic, at the Montreal Medical Chirurgical Society. The patient, a boy 15 years old, had had variola in the winter of 1885–6. He was discharged from the hospital in January, 1886, with ulceration of the right cornea, otherwise well. The attack of chorea began in March, two months after the discharge, and has continued since. He never had paralysis, rheumatism or any cardiac trouble, and his general health is now good. When asleep the choreiform movements ceased, and he exercised a certain amount of control over them at will. Pressure over the median nerve near the elbow controlled the movements. He attended the public school from eight in the morning till five in the afternoon.

Treatment of Chorea.—Owen[108] noticed a case of chorea treated with 4½ minims of Fowler's solution three times daily (the dose afterward increased to 5 minims). One month after the commencement of the arsenic, bronzing of the nipples, armpits and neck occurred; this became more marked during one month in some of the parts affected and then gradually diminished. Cases of bronzing following the use of arsenic are not common. Frühwald, of Vienna, has treated cases of chorea with arsenic administered hypodermically and by the mouth, and compared the results. He gave one to three minims of Fowler's solution hypodermically, and considered that the results were more favorable than dosage by the mouth. Joffroy has had good results in the treatment of chorea with chloral. He gave to children of ten years one drachm daily, giving 45 to 60 grains daily for two weeks or two months. Free cured two cases of chorea, which had been unsuccessfully treated by other drugs, with cimicifuga. Riess,[109] of Berlin, has recently warmly recommended hypodermic injections of physostigmine in chorea, and claims that the duration of the disease is thereby shortened to only fifteen days on an average, reckoning from the beginning of the treatment. In a few cases the choreic symptoms ceased in five or six days. One milligramme (1-64 grain) of eserine sulphate was injected twice a day, and a strengthening treatment generally was adopted. The injections were found useless in very severe forms of chorea with a fatal tendency, also in cases of habitual chorea in adults. Among forty cases spoken of by Riess, four belonged to the very severe forms; the rest were "perfectly cured" by physostigmine. This success induced him to try the influence of this drug in other hyperkineses, and partial success was obtained in tetanus, tremors (senile, alcoholic, hysteric and typhoid), paralysis agitans, multiple sclerosis of the brain and spinal cord, Charcot's post-hemiplegic chorea, and finally in one case of hysteria in the male with extreme emotional excitability, and remarkable increase of all reflex movements. The preparation supplied by Merck, of Darmstadt, was used, but Riess was inclined to recommend the officinal salicylate of the (German) pharmacopœia as being more stable.

Post-mortem in Chorea.—Nauwerck[110] has published the details of a carefully conducted post-mortem in a case of fatal chorea minor, dying of asthenic pneumonia. To the eye no gross change could be detected in the organs after death except a few vegetations on the mitral valve. There was a systolic murmur during life. A histological examination showed that the peripheral nerves were healthy, but in the central nervous system were three kinds of changes. In the medulla oblongata and pons varolii were (1) areas of great peri-vascular infiltration; (2) areas in which were slight hæmorrhages. Both these changes have been described by Dickinson. There was (3) degeneration of the nerve fibres, chiefly in the cervical region of the spinal cord. The change consisted in irregular swelling of the axis-cylinder in parts (the so-called "hypertrophy"), and in fatty degeneration with complete disappearance of the axis-cylinders. These changes were irregularly distributed, and according to Nauwerck, their presence might account for the incoördination.

ATHETOSIS.

Railton[111] describes the case of a girl aged fourteen, who had suffered with infantile hemiplegia, with contractures, since she was eleven months old. She began to walk at three years. At four the contractures in the arm and hand relaxed. Since this occurred, the fingers and thumb and the hand at the wrist have been more or less constantly in that condition of slow movement which is known as athetosis.

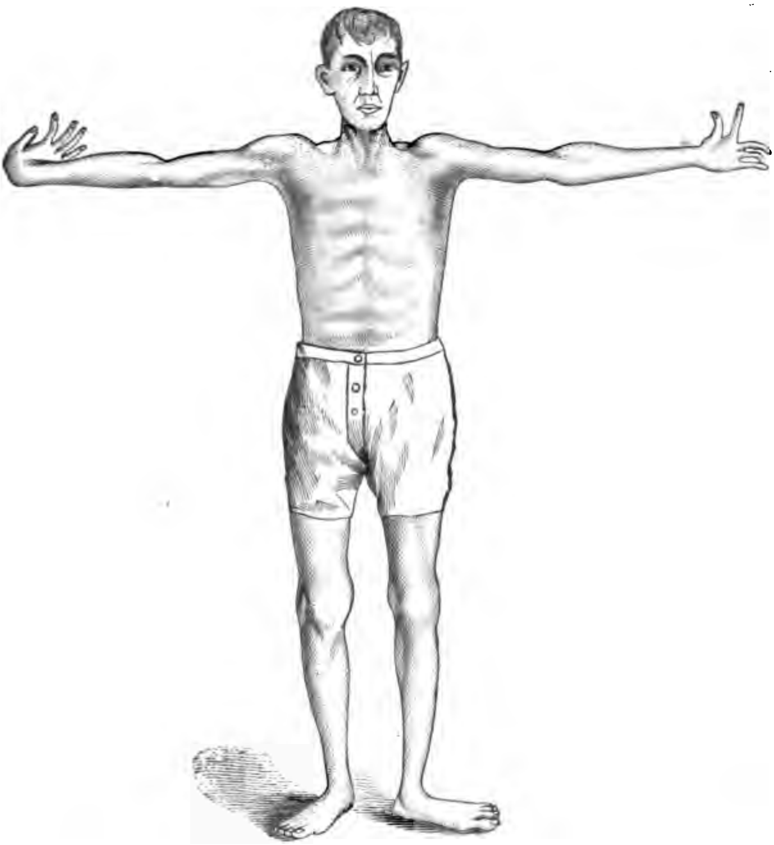
As to the nature of the case:—athetosis is one variety of a group of movements, differing considerably from one another, which are now classed together under the heading of *symptomatic hemichoreas*. They are all the result of some cerebral lesion, in most cases hæmorrhage or softening, but they may also be due to abscess, tumor, injury, syphilis or atrophy from any intra-uterine lesion; in fact they are producible by any lesion of a certain tract of the brain (to which he says he will refer more particularly later on) which so damages, without actually destroying, the motor fibres as to interfere with the regular transmission of impulses, without, however, entirely preventing their passage.

To briefly enumerate these different varieties of movement we have first *hemichorea* proper, in which form there is an incessant shaking of the limbs, especially of the upper extremity. The irregular jerkings resemble the true chorea, and are exaggerated by voluntary movement or by the patient's endeavor to arrest them. The second variety, *athetosis*, an instance of which he was considering, is closely related to the first,—is in fact an attenuated form of it, which not uncommonly succeeds it after a time; and in some cases of hemiplegia the two varieties may be found co-existing, the affected limbs jerking more or less violently during excitement, at other times the hands only being the seat of the slow movements as he described them. The third variety is called *hemi-paralysis agitans*, and in it there is a limited movement of the fingers and thumb in frequent but rhythmical oscillations which persist during repose. The movements become larger during voluntary action, so that the whole limb may be put in motion, in the same way as in true paralysis agitans. In the fourth variety the movements are similar to those of the third except that the oscillations cease when the limb is at rest. This form is called *hemi-sclerosis in patches*. Finally, we have a variety characterized by incoördination during voluntary action, and termed *hemi-ataxia*. It may occur as a symptom without the existence of any involuntary movements, and is probably present to a greater or less extent in the four preceding varieties of movement whenever the cerebral lesion interferes with certain sensory nerve fibres. He summed up his subject by saying that lesions from any of the causes before mentioned, occurring in any part of the motor tract from the cortex above to the commencement of the spinal cord below, may be the cause of any of the varieties of symptomatic hemichorea.

Küssmaul[112] reports the case of a girl 10 years old who had from her birth movements of athetosis involving both sides, hands, fingers, feet and toes. These were usually clonic, but occasionally became tonic. The feet often assumed, in consequence, the position of equino-varus. Other neurotic symptoms were absent. The contractures were not dependent on paralysis of antagonistic muscles, but were an expression of athetosis.

Bilateral Athetosis.—Hughes,[113] of St. Louis, has reported a unique case of bilateral athetosis. This boy had not complete voluntary control over the movements of his muscles; that is, he could not by direct effort of the will, along the regular channels of nerve conduction, restrain either the rhythmical movements or the spasmodic attitude assumed by the fingers; but he could, by strategy, modify both attitude and movement, by bringing one limb to bear upon another and assuming for the affected limbs flexed positions; but no matter how much he succeeded in [illegible] these movements, grotesque attitudes would always be assumed by one or more of the fingers. His affliction unfitted him for any occupation requiring manual dexterity. He tried a number of things, but had to give them up because of physical incompetency.

This young man had no history of epilepsy as it existed in most of Hammond's cases; never had convulsions nor chorea in infancy, nor is there any evidence or history of genuine hemiplegia. The case appears to be in every way a distinctive symptomatic one, even more fully sustaining Hammond's claim for this disease as a distinct pathological entity than this author's own recorded cases.



Bilateral Athetosis.– (Hughes.)
(*Weekly Med. Review.*)

The following is briefly the history of this case:—

George E. M., aged 20, American born, about nine years ago met with an accident on the railroad, causing injury by concussion and direct violence. He was taken from the wreck in an unconscious state, but remained so for a few minutes only. He had two ribs broken, and was hurt in the lower dorsal region, but not seriously enough to affect the functions of bladder, kidneys, or bowels, or the power of moving the lower limbs. He was able to go out within six weeks after the accident, but had an incomplete brachial monoplegia.

About one year after the accident contracture of the left forearm on the breast appeared, and embarrassed movement of the right arm set in, so that he could not throw a stone easily or project it far from him. He "threw like a woman," he says, and could not make a full rotary movement. About four years ago, long after he had fully recovered the general use of the upper extremities, irregular contractions first appeared in the fingers and flexors and extensors of the arms and forearms. About one year ago the left arm grew worse, and the right arm became useless for three months and had to be carried in a sling.

SNAPPING FINGER (DOIGT À RESSORT).

Eulenburg[114] reports two cases of this interesting affection. One of these was a man who was affected in the ring finger of both hands. The cause was supposed to be the pressure of his gun upon the tissues at the base of the finger. An induration was caused in the tendon, which being alternately held and suddenly released during flexion and extension caused a sudden jerking movement. The second case was a compositor, in whom both thumbs and both little fingers were affected. The phenomena were similar to those in the first case. Eulenburg directs treatment to (1) reducing the induration, (2) to widening the sheath. The first indication is met by baths, poultices, iodine, compresses, massage and electricity; the second by fixing the fingers midway between flexion and extension, in order to widen the sheath by prolonged pressure of the indurated part. This treatment is painful. It may be combined with massage, local beating, and electricity. He does not recommend surgical interference.

Mesinger[115] reports a case which he calls "snapping finger" and describes as follows: The thumb of the left hand of a workingman was jerked or snapped, evidently in the inter-phalangeal joint in both flexing and extending. A little protuberance could be felt on the tendon which was held by the narrow sheath, thus causing a snapping or jerking movement. The sheath was cut in the region of the swelling, which was pared down, and the wound sutured. After healing, the cure was complete.

PARAMYOCLONUS MULTIPLEX.

Kowalewski[119] reports a case of this novel neurosis in a woman aged 34, after a nervous shock. The symptoms were symmetrical, tic-like, and even tetaniform convulsions of whole muscles in the extremities, back and neck. The facial muscles were not affected. The intensity and extension of the convulsive phenomena varied, so that there were good and bad days. They were weakened during voluntary power and ceased during sleep. Patient had also giddiness, anxiety and weakness of memory. The reflexes were increased. After three weeks' treatment by galvanism of the cord and sympathetic system, and cauterization along the column, with nourishing diet, a cure was accomplished. He regards grave psychical shock in the neuropathic constitution as the most important etiological factor in these cases. He also thinks that the disease is not so rare as it is sometimes considered, but that it may have been confounded with tic convulsion or chorea major. He regards the symmetry of the affected muscles and the exemption of the face-muscles as pathognostic. Starr[118] described another case of this affection, with valuable comments.

NERVOUS COUGH.

Bresgen[120] makes the following observations: No local cause exists for these nervous coughs; no local treatment, however careful, has been able to cure them. The psychic treatment must be enforced. Rosenbach believes that the cough can always be traced back to a local cause, which disappearing leaves this cough. It is probable that these local causes, coexisting with a weakened state of the general health, are the real cause of the affection. Hence in treatment attention must be paid to the general health, especially to warmth, to bathing, etc.

THE PATHOLOGY OF DISEASES OF THE NERVOUS SYSTEM FOLLOWING MALARIA.

Singer[121] says that neuralgia, paralysis and aphasic affections (?) are the most common disorders due to this cause. The typical appearance, their disappearance with decrease of the fever, and the favorable influence of quinine prove their connection with malaria. Disorders of the central nervous system, as paraplegia, acute ataxia, or of the peripheral system, as neuritis or polyneuritis, are less noted after malaria. When they do appear, it is apt to be in the period of convalescence. He gives a case of acute polyneuritis following severe malarial infection in a man æt. 26. He contracted in Singapore what appears to have been a malarial remittent fever, not amenable to quinine, but which was cured by a resort to a high altitude. Returning to Singapore, he had paræsthesia in the toes, followed by complete paraplegia. At the same time sensory and motor disturbance appeared in the arms, followed by facial paralysis on both sides. There was no fever, no cerebral phenomena, and no affection of sight and hearing. Improvement took place under galvanism, sulphur baths, iodide of iron, and a journey through the Desert, until he had almost recovered when seen by Singer.

WAKING NUMBNESS.

Aulde[122] reports four cases which had come under his own observation. A gentleman in the vigor of manhood, experienced when waking in the morning, or, in fact when waking out of sleep at any hour of the day, numbness and tingling which lasted for a minute or less, but after which everything seemed to adjust itself. In the second case the patient suffered from numbness and tingling in the fingers, so that she was unable to fasten her clothing in the morning, and it was afternoon before she was able to pick up a pin or a needle. The third case was a widow, aged 47, who believed she was threatened with paralysis, having suffered for some time with this peculiar sensation of dumbness and tingling. The fourth case was a married lady, aged 40, who after quinsy requested him to advise her concerning an unpleasant sensation which she had experienced for several years. A numbness, tingling and pricking sensation in the fingers of both hands, made its appearance at almost any time in the day, sometimes at night when she awoke, and occasionally in the morning. Dr. Aulde for his cases gave iodide of potassium and carbonate of ammonia in combination. Dr. Mitchell[123] speaking of this class of cases, says that the disorder may be a mere tingling or actual loss, or lessening of tactile sensation; but in any case it rapidly fades away or yields to a little friction. Dr. Andrew H. Smith[124] describes, under this title, four cases, from a study of which it appears that the numbness is something added to the normal condition. It is a purely subjective symptom. Dr. C. L. Dodge[125] adds two cases of this description.

HEREDITARY TREMOR.

At a meeting of the Medical Society of London, West[126] showed a case of hereditary tremor in a man aged thirty, in whom the fine movements of the hand were first noticed at the age of eight years. The father was "shaky" and died at the age of 58 of paralysis. The tongue and ocular muscles were not affected. The tremors had the character of those seen in delirium tremens. The case was one of the same group shown by Dr. West last year. Dr. Hadden said the handwriting was a fair copy, and did not show signs of tremor. Dr. Herringham alluded to the tremors that occurred in the hand after carrying heavy weights, and thought there was a resemblance to those seen in the man. Mr. Hill believed that the tremors were akin to those seen at the onset of the hypnotic state when artificially induced. Dr. Hughlings-Jackson thought the cases described formed a group of family nervous diseases, other groups being Friedreich's disease, pseudo-hypertrophic paralysis, etc. Dana[127] has published a valuable paper on the same subject, with reports of cases.

CLINICAL STUDY OF THE NERVES OF THE FACE.

Contrary to the opinion of Liebermeister, who holds that in rheumatic facial paralysis its lesion is located upon the facial at its exit from the stylo-mastoid foramen, Prof. K. Ketli,[128] of Buda-Pesth, believes that the lesion is invariably to be found in the aqueduct of Fallopius. The part of the nerve passing through the petrous portion of the temporal bone is easily subject to cold because of the tympanic membrane and thin layer of bone separating it from the outer air. The fact of facial paralysis due to cold being limited to one side is readily explained because only that side is affected by the cold which exposes the tympanum directly to its influence. Again, a slight exudation into the Fallopian aqueduct readily strangles the nerve, which would not be possible in other parts of its course. The following observation is also of great value in the settlement of the still open question as to which is the taste and which the tactile nerve of the tip of the tongue. A young woman of 22 years developed a right abducens and facial paralysis, without impairment of taste, and after a time anæsthesia in the area of the second branch of the trigeminus, coincident with a loss of taste in the right anterior half of the tongue, while the tactile and temperature senses remained normal. Several weeks later the anæsthesia extended to the third branch of the trigeminus, and at the same time the right anterior half of the tongue lost its tactile and temperature sense. It is rational to conclude that the nerve of sense for the anterior third of the tongue is the chorda tympani, and that the tactile and temperature nerve for the same region is derived from the lingual, the former originating from the spheno-palatine ganglion (Meckel's) of the second division of the fifth, and the latter from the third division of the fifth.

WRITER'S CRAMP AND IMPAIRED WRITING POWER.

Poore[129] read before the Royal Medical and Chirurgical Society a paper (which was a continuation of one published in the sixty-first volume of the Society Transactions) entitled "An Analysis of 93 Cases of Writer's Cramp and Impaired Writing Power, making with 75 cases previously reported, a total of 168 cases. The cases as reported were given in the form of tables, as an appendix to the paper. They were arranged in three groups: (1) paralytic, 13 cases (10 cerebral); (2) degenerative, 14 cases; (3) neuro-muscular, 66 cases. The cases in groups 1 and 2 were nearly all referable to well understood pathological conditions which affected the brain or spinal cord. The author was of the opinion that a not uncommon cause of impaired writing power was to be found in lesions causing slight degrees of a hemiplegic type, and that the evidence of a slight, long antecedent, and possibly forgotten hemiplegia might have almost disappeared, except in the execution of an act such as writing, which required both delicacy and steadiness. Slight degrees of sclerosis affecting the different columns of the spinal cord might in like manner be scarcely noticeable, except in the effect which they produced on the act of writing. Of the total 168 cases of impaired writing power, about one-fourth were referred to slight central changes in the brain or cord. The remaining 177 cases (66 in the present paper and 51 in the previous paper) could not with any sufficient reason be referred to central change. They formed a group which the author called the neuro-muscular group. The objective symptoms of this group, in addition to the impaired writing power were: (1) nerve-tenderness; (2) change in the faradic irritability of some of the muscles; (3) tremors. These symptoms might occur singly or combined. Nerve-tenderness occurred in 50 out of 93 cases, and in most of these it was the median nerve which was tender. Change in muscular irritability occurred 61 times in the 93 cases. The change was generally a depression, occurring chiefly in those muscles which, in the act of writing, were subjected to prolonged contraction, such as the interossei, etc. Tremor occurred in more than half the cases. Many cases were complicated by slight rheumatic conditions of some of the joints. In the treatment of cases of impaired writing power, the first step was clearly, diagnosis. For cases of the neuro-muscular group, blistering and other forms of counter-irritation, applied to the tender nerve trunks, had proved of marked value. Good results had also been obtained from massage and other methods of improving the nutrition of the affected limb.

NERVOUS CARDIAC DEBILITY (NEURASTHENIA-VASOMOTORIA).

Rosenbach[130] would indicate by the above term pure neurosis of the heart, without any organic change, which commonly obtains as a part of the phenomena of general neurasthenia. It is an affection of early adolescence and of puberty, and mostly of the anæmic or nervous. Severe bodily or mental exercise, abuse of alcohol, coffee, tobacco, etc., and violent emotional excitement, are etiological factors. The affection exhibits two well defined stages,—one of excitement and one of depression. The first is characterized by rapid changing congestion of the hands and face, by paræsthesia of the chest and upper extremities, and hyperæsthesia of the cardiac region, in consequence of which anxiety and palpitation occur without any appreciable cause. Often insomnia, anorexia, constipation, and frequent micturition are present. In the second stage, the patient suffers from continued paleness, psychical depression and lassitude. Palpitation and arterial pulsation are felt in spite of lowered heart power and weak pulse. Reflex excitability is enormously increased and the patient suffers from headache, dizziness, faintness, anorexia, etc. Together with hygienic and dietetic treatment, bromide preparations, iron, quinine, ergotin, and especially cocaine (half a grain twice daily) are useful.

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DISEASES OF THE HEART AND PERICARDIUM.

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PERICARDITIS.

Etiology.—Duroziez[1] affirms that rheumatism is the most frequent cause of pericarditis as well as endocarditis, and Hennock[2] has lately recorded cases in which the pericardial inflammation preceded the articular manifestations. Beyond this, he considers tuberculosis and pleurisy as among the chief causes, particularly in young subjects. According to Gerhardt, pericarditis is more frequently a complication of scarlatina than is endocarditis, as stated by Hennock; but he agrees with Hennock that in children the pericardial change precedes the rheumatic manifestations. Jaccoud[3] relates a case bearing upon this point, in which scarlatina caused cardiac hypertrophy and myocarditis in a patient of seventeen, who, ten months later, was attacked by acute articular rheumatism complicated by endopericarditis. Diphtheria and erysipelas are both named as causes, and Champiel[4] reports a case of pericarditis associated with latent tuberculosis in which dyspnœa was the only symptom. An osteomyelitis is stated by Broca[5] to have caused endopericarditis, and Da Costa[6] reports a case apparently of idiopathic origin.

Symptoms.—Potain[7] has determined that temperature is of very little value in determining the accession of pericarditis in acute rheumatism. An abnormally low temperature has been noted in a few cases—a sort of algid condition—at the outset of the pericarditis. Pain, according to Sibson, is present in only 70 per cent. of cases, and is often very slight. It may be a sense of weight, or constriction over the præcordial space, or become intense and lancinating, extending to the back or arm. Under all circumstances it is increased by pressure over the heart, and especially by upward pressure under the free border of the ribs (at the left of the median line). Hyperæsthesia is quite frequently present. In rare cases it follows the course of the phrenic nerve, or may assume the form of rheumatismal angina,—a condition which renders the prognosis exceedingly unfavorable. Dyspnœa during its early stage may be due to the irregular action of the heart caused by the primary inflammatory irritation or to the intensity of the pain. Later it is caused by the effusion and consequent heart failure. Dysphagia has been noted in three cases. The pulse is generally accelerated. It is called "paradoxal" when the pulse wave is diminished during inspiration.

Irregularity of the heart's contractions only occurs when there is much effusion; and then pressure on the auricles may distend the jugulars. The venous pulse proper does not occur in pericarditis unless there is a valvular complication.

Eroding friction sounds are developed during the first two days. They may often be felt on palpation, and vary from the softest "frou-frou" to a harsh scratching. Friction sounds are not usually coincident with the apex beat, but mesosystolic or mesodiastolic, commencing after the normal sounds. To the ear the sound is always superficial, seeming to be between the ear and the chest wall. Potain objects to Gubler's description of the friction sounds, for the latter implies that they precede or follow them, which he does not think the case. He also objects to Sibson's remark that they come by the side of them. But he endorses the latter's observation when he says that they do not begin with an "accent," which shows that he had noted the relation of all valvular sounds to the slight interval. Potain uses the designation mesosystolic, mesodiastolic or presystolic in describing each particular case. It is the presystolic friction as he hears it that gives the gallop rhythm. He rejects Sibson's explanation of the lack of synchronism between the normal heart sounds and the friction sounds. His error he supposes to have consisted in confounding the muscle contraction with the movements of the heart's surface. The latter depends on the change of the heart's form and volume, which is progressive. Friction sounds are usually heard loudest or only during inspiration. Their usual site is about the third intercostal space, sometimes at the base, rarely at the apex. They are not propagated, are greatest when the patient is sitting, and are increased by pressure. A gallop rhythm is heard sometimes at the outset of pericarditis. Potain believes that it is due to the fact that the myocardium has lost its tonicity and allows the ventricle to be filled abruptly. This causes a shock, together with the sound preceding the normal sounds, and so making up the gallop rhythm that is premonitory of pericarditis, and may precede all friction sounds. The gallop rhythm of interstitial nephritis is similar, but more intense. Right heart dilatation has also a gallop rhythm whose origin is a gastro-intestinal reflex; but its point of maximum intensity corresponds to the right heart and it is transitory and without fever. The gallop rhythm may appear at the very commencement of the rheumatism or during the second week.

Præcordial Dullness.—The pericardium artificially injected in the cadaver was found to assume the "encoche" form (that of the natural loaf of bread), which corresponded precisely with the form of the dullness noted on the 21st day in the case which Potain reports. Also the area of pericardial dullness in this case had become doubled within the five days of maximum effusion, while the area of uncovered pericardium was increased eightfold,—a state of things which could not occur in simple dilatation.

The displacement of the visible apex beat upward and to the left, toward the second intercostal space, is often rapid, depending of course upon the rapidity of the effusion. As the lower line of the effusion dullness becomes depressed, the distance between the two increases even more rapidly than the apex rises. Bulging of the præcordial area is a symptom of little value, as it occurs only in children with yielding chests. Weakening of the heart sounds takes places most rapidly at the apex, and varies with the amount of the effusion and the extent of myocardial changes. Dilatation may follow an extension of the inflammation to the myocardium, or through myocardial changes induced by pressure of persistent and extensive effusion, in which case it is liable to become permanent. Pericardial adhesions often become the most serious sequelæ of the process. When partial, they may cause irregular heart action, particularly upon exertion, and may lead to degeneration of the cardiac muscle. When complete, they may even be the cause of death. Bard and Tellier,[8] however, report a case in which two layers of adipose tissue covering the ventricles allowed a re-establishment and maintenance of the heart's motion even with complete pericardial adhesions. As illustrations, the following clinical reports may be cited:—

Babcock,[9] of Chicago, reports a typical case, which had suffered, six years before, with acute rheumatism for a year. The symptoms included præcordial pain, intensified by dorsal decubitus, persistent cough, severe dyspnœa, reflex epigastric pain, with temperature of 102° F. and a pulse of 120. Friction sounds and increased area of dullness were characteristic. Interscapular pain was explained (?) at the autopsy by a large inflammatory area over the posterior surface of the left auricle. Cenka[10] affirms that friction sounds have been audible, even in the presence of a quart of effusion, when there were folds and projections of fibrin on the parietal pericardium. Matray,[11] of Vienna, describes a case in which a sharp whistling note was heard between the fifth and seventh intercostal spaces, loudest near the termination of expiration. The autopsy showed fibrinous bands extending from the cardiac wall to the pericardial sac. These, as Gerhardt, Rosenbach and Lichtheim agreed, were put on the stretch by the pressure of the serious exudation and the retraction of the left lung.

Diagnosis.—Potain[12] observes that the diagnosis is easy when one has been able to observe the grazing sounds developing into the friction sounds of pericarditis, but not so easy when one is called in at a later stage of the disease. Tyson[13] says that the ordinary cardial area is about 2½ inches square, and that if the dull area extends up to the third interspace, it indicates hypertrophy or pericardial effusion; and he distinguishes between these by the sudden change from resonance to dullness in the case of the latter, instead of more gradual change in the former.

Bucquoy[14] says that a slight or dry pericarditis at the base of the heart often leads to a mistaken diagnosis. The seat of the friction sound that is apt to be mistaken for a murmur is, he says, in the third or fourth left intercostal space near the sternum, and that, though it may be audible at the apex, you will not often get it there until it has first been detected toward the base. He distinguishes them by their want of synchronism with the systole and by their non-propagation.

ENDOCARDITIS.

Etiology and Pathology.—Pathological research upon the subject of endocarditis has been devoted mainly to the establishment of the etiological relations of various forms of bacteria to acute ulcerative processes. A fair summary of both their own and other researches has been given by Stern and Hirschler,[15] clinical assistants to Prof. Koranyi, of Buda-Pesth. Investigators seem not to be entirely agreed as to the forms of bacteria which may be accidental or casual. Bobes[16] found: (1) a coccus resembling that of erysipelas; (2) one similar to that found in pneumonia; (3) tubercle bacilli; (4) spheroidal and oval micro-organisms' both single and in chains. As a rule, the organisms found in the endocardium were those of the primary disease, of which the endocarditis was a complication.

Wechselbaum[17] and Wyssokovitsch[18] also found various forms of micro-organisms, of which the staphylococcus pyogenes aureus and streptococcus pyogenes were the most prominent. In cases of endocarditis vernicosa, as a rule no bacteria were found. In two cases of endocarditis, where the processes had become reparative, no bacteria were found. Inoculations of animals with each form of coccus produced typical endocarditis. The diplococcus of pneumonia alone also caused ulceration of the endocardium and death in some cases. Ribbut[19] produced endocarditis and myocarditis with the staphylococci without previous injury of the valve, although in most instances wounding of the valves was essential to the induction of ulcerative changes from inoculation. Netter[20] considers Fränkel's pneumococcus as undoubtedly the etiological factor in cases of endocarditis complicating pneumonia.

Stern and Hirschler's own experiments are confirmatory, and they agree with Wechselbaum and Wyssokovitsch that, while the staphylococcus pyogenes aureus and albus and streptococcus pyogenes are the etiological element in most cases, yet other and various forms of micro-organisms may induce the same ulcerative changes. A combination of several forms of cocci was more powerful than any one alone, and their pathogenic action was increased by all forms of depreciated vitality. Wyssokovitsch seems to have determined that the streptococcus grows the faster, causing an anæmic necrosis, and the staphylococcus more slowly, with suppurative changes.

The report of Fränkel and Sänger[21] upon thirteen cases of endocarditis vernicosa seems to advance a step further. In these cases they found eight different micro-organisms, five of which reproduced the disease. As most of the patients had suppurating foci which were apparently the centres of infection, they were led to consider the form as also a mycotic disease. They state that the bacteria were also found in old endocardial products, indicating thus a strong vitality, and possibly explaining recurrent endocarditis. The frequency with which they locate in the left heart is ascribed to their need of oxygen. On the other hand, Rosenbach,[22] of Breslau, believes that all forms of endocarditis yield micro-organisms, but that there is no specific source of infection, and that the necessity of some valve lesion antecedent to their development is only what is well known as regards all mycotic infections. More recently Prudden has repeated in a measure previous experiments and has endeavored to make them thoroughly reliable. The first set were simply examinations to determine the presence of bacteria in various forms of endocardial inflammation. In seven cases of chronic vegetative endocarditis no bacteria were found; and in eight out of thirteen cases of acute ulcerative endocarditis, none were demonstrable even by culture. Clinically the bacterial and non-bacterial cases could not be distinguished. In the second set of experiments, inoculations were made of bacteria derived from a case of acute malignant ulcerative disease, in which autopsy showed ulcerations and abscess of the endocardium. The result of each inoculation after previous injury of the valve was typical ulceration. A third set of control experiments indicated that neither injury of the valve nor inoculation alone was sufficient to induce the ulcerative process. From the fact that most cases of the mycotic form gave evidence of old endocardial disease, he is inclined to consider that chronic inflammatory changes may take the place of the traumatic factor in etiology. Prudden draws attention to the fact that only the streptococcus pyogenes and staphylococcus pyogenes aureus have been found in man, and in connection with the fact that the injection of either in a rabbit may cause death by pyæmia, but does not induce ulcerative endocarditis until there has been traumatic or chemical injury of the endocardium, makes the valuable and important point that the conditions in man and animals may be, and probably are, very different.

Wyssokovitsch emphasizes this same point, insisting that not only may the vitality of the animal be so affected by the operations as to nullify its value, but that their reaction to various forms of organic, as well as inorganic poisons, may so differ from that in man as to greatly lessen the value of such experiments. Upon this point we most decidedly agree with him. Equally important is his admission that, among the numbers of bacteria found in endocardial ulcerations, it is impossible to say which is the specific etiological factor and which the product of the necrotic process.

Prudden concludes: (1) That, while bacteria (generally of the spheroidal type) are present in a certain proportion of cases, yet many cases marked by large formation of thrombi and multiple infarctions, occur, in which no specific bacteria can be found, and that such cases are usually associated with evidences of old inflammatory endocardial changes. (2) That the bacteria which experiments have shown to be causative factors in endocarditis, first find lodgment where endocardial surfaces have been injured; where old inflammatory changes are present or under other unknown conditions. (3) That bacterial ulcerations cannot be recognized by gross appearances. All experimental research goes to show that some predisposition is necessary for the development of bacteria within the endocardium. When the primary lesion is traumatic, inoculation is not successful after about two days. This fact, in connection with the occurrence of ulcerative disease in apparently healthy endocardial tissue, seems to have disturbed some observers. It does not seem at all difficult, however, to believe that a transient traumatism may cause less depression of cellular vitality than chronic interstitial changes or even the chronic traumatism attendant upon high arterial or ventricular tension and similar cardiovascular conditions.

Under the stimulus of pathological research, clinical reports become more and more corroborative of the etiological relations of bacteria.

Houtang[23] records a case of the embolism of popliteal and axillary arteries, dependent upon an endocarditis which was not ulcerative, but followed an application of caustic to a corn. Broca[24] describes an endopericarditis following an osteomyelitis with suppuration, produced by a long walk in tight boots. Stern and Hirschler[25] report a case complicating pneumonia and one resulting in embolism, which was attributed to a urethritis of eight years' duration. Netter collected 82 cases of pneumonia complicated by ulcerative endocarditis. His conclusion that the two conditions were dependent upon a common cause was not admitted by Fraube, although Wechselbaum had found similar cocci in the two diseases. Velden[26] also reports two cases of endocarditis without joint complications, due to gonorrhœa, and Pel,[27] of Amsterdam, one following an ulcer from an ill-fitting shoe, which was associated with multiple embolisms. Augier and Desplats[28] describe a case of recurrent endocarditis, with calcification of old clots at the aortic valves, and fibrinous exudation, with evidences of recent inflammation along the free borders. Spheroidal micro-organisms were found on the calcifications and in the spleen, and a grain of calcareous matter at the apex of an infarction of the kidney.

Kundrat offers the hypothesis of simple mechanical causes, as determining the location of the primary mycotic infection of the endocardium. He claims that the action of the valves upon the blood tends to favor coagulation upon their borders, and that these shreds of fibrin offer suitable resting places for the cocci. In support of this theory, he states that the ribbon-like bands of fibrin found upon the valves often exhibit no signs of inflammation, but are simply shreds of whipped out fibrin. The result of the mycotic theory of endocarditis, when proven, must be to remove the disease from the class of heart diseases to that of general constitutional diseases with local manifestations. At present, however, although it is undoubtedly often a mycolæmia with accidental manifestations in the endocardium, its etiological elements are not sufficiently well established or classified to warrant any change in its classification.

Symptoms.—Little of value has been added to the symptomatology of the disease. In general, reported cases go to show the wide variations in both rational symptoms and physical signs. One case reported by Pel[29] presented a subaortic stenosis from vegetations, causing a murmur which was mistaken for that of pericarditis. One case among those recorded by Stern and Hirschler[30] was marked by chill and fever every fourteen days, with loud systolic apex murmur and enlarged and tender spleen. This was followed by a condition of quotidian intermittent with temperature of 104°. At the end of a month the fever disappeared but the cardiac symptoms remained. The pain and swelling of the joints soon returned, however, with increased fever, a double first sound and later aortic murmur, ecchymoses over the entire body and death from exhaustion. In a second case the symptoms were those of a pericarditis with increased area of cardiac dullness. The special symptoms, in average cases upon which diagnosis must be based are fever: often irregular or intermittent, or remitting for a considerable period without apparent cause; rapid evolution of the cardiac symptoms, often in irregular or inverted order; enlargement and tenderness of spleen; multiple infarctions of joints, liver, kidney, etc. Eichhorst recognizes a typhoid and intermittent form of fever, and Bramwell distinguishes a pyæmic, a cerebral and a cardiac form, while Leyden makes four classes, as distinguished by the fever.

Prognosis.—Rosenbach[31] states that the dangers are not determined by the presence of bacteria or the fact that ulcerations have attacked the valves, but the circumstance of the patient being a pyæmic subject. Several cases of recovery are reported, although the fact of recovery has suggested the possibility of an error in diagnosis. Stern and Hirschler[32] report a case of recovery after the development of extensive ecchymoses.

Treatment.—Stern and Hirschler, representing the authority of Prof. Koranyi, state that no remedies employed have had any effect upon the pathological processes, and that the treatment must be purely symptomatic. They have given corrosive sublimate both hypodermically and by inhalation, but with no results. Other observers report cases of recovery, but with no special connection with the remedies given. McClun,[33] however, reports a case treated by inhalations of carbolate of iodine, in which improvement was rapid and apparently due to the inhalations.

VALVULAR DISEASES.

Etiology.—The rôle of nervous influence in determining cardiac changes, both muscular and valvular, is discussed in its general relations by Mills,[34] of Canada, who advances the theory of a constant trophic nerve force, which he considers as of vital importance in determining degeneration and destructive changes in the cardiac tissues. In this line, Teissier[35] reports case of perforated aortic valves in connection with locomotor ataxia, in which he considers the change to be a genuine trophic lesion. Leyden[36] considered it very difficult to demonstrate any connection between tabes and valvular lesions, even when the relations between central nervous disease and functional cardiac disturbances are distinct. Still further, Schnell[37] denies any direct relations of cause and effect, though he admits that bulbar paralysis, goitre, and mental emotions even, may indirectly cause organic disease of the heart through a sequence of disturbance of nerve cells, palpitation and nutritive lesions of the myocardium, ending in either hypertrophy or dilatation; neuralgic affections of the left arm, he thinks, act especially upon heart nutrition by the relations of anastamosing fibres from the cervical sympathetic. It is not impossible that the accepted etiological relation of chorea to valvular disease may find a neuro-trophic explanation.

Mitral stenosis appears to be very frequently of so-called idiopathic origin, and when pure and simple is found almost exclusively in women and children. Duroziez[38] gives a list of cases of "mitral stenosis, pure and simple," and divides them into (1) cases where no history of disease can be found, occurring in children who could not run; and (2) cases traceable to disease. Under these last he mentions pregnancy in its relation to mitral stenosis, and states that out of twenty-six cases suffering from stenosis, only four died during pregnancy. Of the specific fevers, typhoid acts upon the muscular rather than valvular parts of the heart; two cases he attributes to chorea, and fourteen to saturnine poisoning, with syphilis.

In a later article[39] he attempts to prove that pure mitral stenosis is not of rheumatic origin, and records eight cases of children who could "never run," but who were the subjects of acute rheumatism in later life. He concludes that "pure" mitral stenosis (infantile or congenital) should be carefully distinguished from that due to disease; a double second sound is not sufficient for a diagnosis and pure mitral stenosis remains "pure" after the patient has gone through several attacks of articular rheumatism. Landouzy[40] finds pure mitral stenosis only in women, and Schnell reports cases to show that funnel stenosis is always pure, and never rheumatic. In a later article[41] he calls attention to the dependence of certain cardio-vascular conditions upon gastric disease and instances two cases. He considers the cardiac disturbance to be reflex through the vagus. It may be mentioned that Guitéras[42] presents some cases to show that an aortic regurgitation may be the cause of a mitral presystolic murmur, by driving or floating out the mitral curtains into the current of blood from the auricle. He considers it possibly the same as Flint's functional presystolic murmur. It is very doubtful if a presystolic murmur can be thus formed. If the aortic regurgitant current enters the ventricle with more volume than the auricular current, such a murmur might be possible, but under these circumstances the filling of the ventricle would be but slightly from the auricular blood, and the circulation would become almost *nil.*

The report of Dr. Neve, of Bombay, Corresponding Editor of the ANNUAL, is of special interest as bearing upon the etiological relation of high altitudes and syphilis to organic cardiac and arterial disease. He states that he has not found forty cases of organic heart disease or aneurism in forty thousand cases seen during his ten years' residence in the East, although syphilis is widespread and often very severe in its manifestations. Such a remarkable condition he ascribes to the exceedingly quiet mode of life and freedom from exertion, although he mentions porterage duty over high mountain passes as among the occupations of some natives. The apparent and hitherto unquestioned relation of syphilis to organic disease and valvular lesions of the heart in this country, when compared with the above statements, suggests the inquiry whether occupation has received sufficient credit as an etiological factor. Increased blood tension in the left ventricle is already recognized as a cause of mitral disease, and Dr. Neve's report should call attention to all conditions causing increased aortic tension as possible causes of aortic valvular disease. Potheret reports a case of aortic insufficiency without stenosis, which perfectly illustrates this condition and the possibility of its occurrence. A similar relation between conditions causing increased arterial tension and mitral disease is noticed by Sibileau.[11] A list of seventeen cases of abdominal tumors associated with mitral insufficiency suggest intra-abdominal pressure (from disease of the kidney, tumors, etc.) as a condition which may lead to mitral insufficiency without change in the aortic nerves through hypertrophy and dilatation. Any other cause of hypertrophy and dilatation may, of course, act in a similar manner, so that (see symptoms) capillary spasm, blood conditions, etc., must be included. Perhaps no one has done so much to make clear the relations between primary blood poisoning from excrementitious products and secondary changes in the heart as Fothergill.[44] He ascribes the primary trouble to old time "biliousness" or perverted metabolism in the liver. Whether due to over-feeding and demands upon the liver function in excess of its capacity, or an inherently weak liver which is unable to cope with even a reasonable amount of nitrogenized matter, the results are the same, an excess of uric acid and other partially converted waste products in the blood, causing at first capillary spasm and overaction of the heart in maintaining the circulation. Such conditions cause heart failure, which may be primary but is more frequently secondary to some valvular lesion, either aortic or mitral. Upon this basis a possible explanation of the remarkable facts set forth in Dr. Neve's report, may be found in the fact that the basis of diet in India is found in rice and other starchy foods almost to the exclusion of animal food. Huchard recognizes a similar condition in dividing heart diseases in three classes due primarily to (1) the arteries, (2) the valves, and (3) the muscular wall.

Pathology.—A very sharp attack has been made, or rather renewed, by Dr. Dickinson[45] upon the generally accepted theory of the formation of the "presystolic murmur so-called," which is indicative of mitral stenosis. He is supported by McVail, Taylor, Harley (whose short note indicates that he for one, at least, is possessed of some of the *impenetrabilia* which he mentions), Travis, Turner, Hanford and others.[46] The defense has been under the leadership of Dr. Gairdner,[47] who had previously offered most valuable contributions to the pathology of cardiac murmurs. He has the assistance of Graham Steele, Balfour, Bristowe, Sainsbury, and many others.[48]

Dickinson's article is but a renewal and clear statement of the opinions of Dr. Barclay, advanced some fourteen or fifteen years since. Dr. Dickinson's arguments may be briefly stated: (1) The weak auricle cannot have sufficient power to produce the loudest, harshest of all the murmurs. (2) When accurately timed, the murmur will be found to be synchronous with the earliest indications of systolic contraction, though not with the first sound, which is short, sharp and valvular in character, and indicates the closure of the stiffened mitral valve by a regurgitant current that has been passing the mitral orifice during the primary systolic contraction. (3) The so-called presystolic murmur runs up and into the regurgitant murmur when that last is present, with no appreciable pause between them, which could not happen if there was a reversal of the current. Dr. Dickinson would thus regard the murmur as covering the first portion of the systole up to the point of closure of the valve and prefers to call it an "early systolic murmur." Dr. Harley's support of Dr. Dickinson appears to have consisted in denunciation of those who were so obtuse as not to accept the opinion of Dr. Dickinson, and is therefore of no value. Dr. Taylor regarded the murmur as produced among the chordæ and not at the orifice. Dr. Travis laid special stress upon the stiffness of the valves and the extra or ventricular force required to close them in diseased conditions. In reply Dr. Gairdner repeated more or less his previous arguments, laying special stress upon those cases in which a mitral murmur begins immediately after the second sound and covers the entire diastolic period. He also referred again to his unique case of ball valve obstruction of the tricuspid. As to the blending of the presystolic and systolic bruits, he plainly indicated that it was never so complete as to present differentiation with proper care. In addition, Dr. Sainsbury recalled the hypertrophy of the auricle, indicating that it certainly takes an active part in propelling the blood in cases of mitral stenosis, while Dr. Balfour emphasized the element of increased tension in the pulmonary circulation. Dr. Sheaver also instanced the *bruit de diable* as proof that the loudness of a murmur was no exact indication of the force of the blood stream. Dr. Bristowe's paper,[49] however, was the most complete and unassailable. He first describes three possible mitral diastolic murmurs which may in proper cases be distinctly recognized, and after discussing the relations of second sound, thrill and mitral regurgitant murmurs, to mitral obstruction, answers directly the arguments of Dr. Dickinson. Dr. Bristowe's arguments in favor of the presystolic nature of the murmur are: (1) that it accords with the physiological fact of the auricular contraction *running into* the ventricular systole, the beginning of which is indicated by the very sound that terminates the murmur; (2) the murmur precedes the pulse and ends with the apex beat; (3) the presystolic murmur does not differ in quality from the earlier mitral murmurs which are acknowledged to be direct; (4) all the direct mitral murmurs are characterized by consonatal noisiness and lack of penetrating power, and are audible over a curiously restricted area, but are not conveyed into the axilla as all regurgitant mitral murmurs are. One argument of Dr. Bristowe seems unanswerable: that if the mitral murmur is a regurgitant one, due to prolonged contraction of the ventricle, there then ought to be, in those cases which also have aortic obstruction, a presystolic aortic obstructive murmur,—systole in this case being understood as the closure of the mitral valve. We have no hesitancy in expressing our own opinion as decided upon the side of the generally accepted interpretation of the origin of the murmur indicative of mitral stenosis. Had we been undecided, Dr. Bristowe's paper would have proved convincing.

Primary valvular lesions are so constantly upon the left side that the possibility of their occurrence in the right heart is often forgotten. Malibran[50] reports a case of primary tricuspid insufficiency, with usual signs of tricuspid regurgitation and a distinct galloping murmur on level with apex, soft and blowing in character. There was no left heart lesion and the patient died from disease of the kidney.

Dr. Porter[51] has also reported a case of extensive right side disease with only aortic stenosis on the left, in which the primary changes seemed to have been in the right heart. Dr. Porter states that in two thousand necropsies, only two cases have been found giving evidence of primary right heart disease. A distinct case of primary tricuspid incompetence from endocarditis is also reported.[52] Such isolated reports thus specially emphasize the fact that physical signs of primary pulmonary or tricuspid disease must be questioned most closely, even while calling attention to the possibility of primary right heart disease.

The statement of Guitéras that pulmonary murmurs are the most frequent of all cardiac murmurs, seems to the writer incorrect. These so-called pulmonary murmurs are undoubtedly extra-cardial and respiratory. Indeed, Guitéras' own statement that they can be produced in most persons by respiratory changes, seems to show that they are not cardiac in their origin. His explanation of respiratory movements causing decreased tension and a consequent eddying in the pulmonary currents, can hardly be accepted. In the majority of cases they will be found to disappear with the cessation of respiration.

Jaccoud,[53] Potheret,[54] Le Guen,[55] and others report cases representing extreme conditions of pathological change in valvular disease, but illustrating no specially new process. Several instances[56] of extreme calcification of the valves show the extent to which pathological changes may go without entirely destroying the functional power of the heart. The intimate relations between valvular disease and general arterial sclerosis is also illustrated in Potheret's report. Rosenbach[57] claims to have shown that in aortic regurgitation, in proportion to the extent of the lesion, the regurgitant current will put greater or less tension upon the capillary muscle and apex, which in some cases may prove sufficient to destroy a previously existing mitral obstructive murmur. We are inclined to doubt such an occurrence, as the mitral murmur is not caused by the falling out of the valve, but by thickening adhesions or vegetations, which cannot under any circumstances be removed entirely from the course of the blood current. In direct opposition is the statement of Guitéras[42], that aortic regurgitation lifts the mitral flaps into the blood current and causes a mitral presystolic murmur.

Dr. Hochsinger[58] reports a case and quotes one of Drozda, in which a perforated aortic valve offered an anatomical explanation of musical murmurs. He can hardly intend, however, even to suggest that such a condition is always present with musical heart murmurs. Matray[59] reports a case where filamentous attachment between the heart and pericardium caused a loud musical systolic murmur, attributable, as he believes, to sudden tightening of this band. Under the failing heart's action just preceding death, the musical note disappeared. We believe that very many musical murmurs are of extra-cardial origin, and in every case the cause is a pathological curiosity rather than a diagnostic or prognostic point of interest. Litten[60] explains the absence of murmurs in certain cases of aortic insufficiency by the low tension in the artery not being sufficient to cause sufficient vibration for sound. He does not mention the probability of the pressure of aortic dilatation in such cases. We are inclined to think such a condition present in these cases, and that careful auscultation will reveal a murmur in the artery when none can be heard over the valves.

The possibility of spontaneous cure of a valvular lesion is illustrated (?) by a reported case[61] in which one leaf of the aortic valve had enlarged and filled a gap made by contraction of the other two leaves.

Symptoms and Diagnosis.—Neukirch[62] (Nürnberg) states that double second sounds are purely physiological and may be distinguished from a presystolic murmur by (1) The duller character of the latter. (2) The longer pause before the murmur. (3) The murmur is loudest over ventricles, the double sound over vessels. (4) One or the other element of the double sound is weaker, as one listens over aortic or pulmonic valves. (5) The murmur is more constant, while a double second sound indicates only inequality of tension in the pulmonary and aortic circulations; a double first sound implies serious change in the muscles as well as valves. As a basis for determining the earliest indications of murmur, L'Huillier[63] states that the tone quality of the normal heart sound is represented by sol–si, a quality which is determined by the diameters of the orifices, and that a change in this musical character of the sounds may indicate disease before the advent of the classical murmur. A list of seventy-one cases,[64] of which sixty-five were in women, give basis for the statement that tricuspid stenosis is never found unassociated with mitral stenosis. Malibran,[65] however, reports a case of primary tricuspid incompetence due to endocarditis. The exceeding rarity of such cases renders them of slight clinical interest. That an organic murmur at the mitral, as stated by Sibileau,[66] may be heard as high up as the second costal cartilage, and Duroziez's affirmation, confirmed by Rosenbach,[67] that venous diastolic murmurs often simulate aortic regurgitation, illustrate the intricacy of cardiac diagnosis, which depends alone upon auscultation. Prof. Stiller[68] makes a similar statement as to the strangely latent and obscure nature of many valvular lesions. He lays special stress upon the necessity for both anatomical (physical) and physiological diagnosis, and places hypertrophy of the liver as the earliest sign, not excepting dyspnœa, saying that percussion is of more value than auscultation.

He holds that a functional mitral insufficiency may really account for an anæmic murmur, which is supported by the fact that it is mostly audible at the base of the heart. He ascribes also early ascites in cardiac disease to the liver change, and notes the fact that poor patients often recover from dropsy more readily than rich, because in such cases it does not so certainly indicate cardiac failure. Finally he notes those cases in which the sequelæ alone indicate a latent heart disease. Rondot[69] contributes an article upon the semi-lunar space and its modifications due to disease of the heart, lungs, stomach, liver, spleen, etc. The name is applied to the space on the left anterior wall of thorax giving semi-tympanitic percussion and indicating the location of upper portion of stomach. Rondot considers this space as of special value in determining pathological changes in the heart. Normal modifications of the space are caused by digestion and respiration. Pleuritic effusion presses a triangular area of dullness into this space or carries the gastro-cardiac line to the right. When absorption occurs, the left border of the space may be carried beyond normal. With pleuritic adhesions the semi-lunar space may persist even during marked effusion. The cardiac conditions affecting this space are: (1) pericardial effusion; (2) dilatation and hypertrophy; (3) displacement of the heart. It is to be noticed that when the heart alone is causing decrease in semi-lunar space, the area of pulmonary resonance to the left remains intact.

Certain variations in valvular murmur from accepted rules of diagnosis are noted by Steele.[70] He notes particularly the non-conveyance of a mitral murmur to the back as being associated with weak and dilated heart, which makes but a feeble murmur, although all the rational signs of severe mitral regurgitation and obstructed circulation are present. A similar condition is found in hearts where the dilatation has resulted in mitral incompetency even while the valves are healthy. Among the modified heart sounds which are at times difficult of interpretation is the so-called gallop rhythm,—a peculiar triple sound. Cuffer and Barbillion[71] have given a full description of its formation by the combination of one abnormal with the two normal heart sounds, from which may be formed a diastolic or mesosystolic gallop, the abnormal sound in the diastolic just preceding and the mesosystolic immediately following the cardiac impulse. When permanent in the left heart, the diastolic gallop indicates hypertrophy, and the extra beat, which is presystolic, is said to be a bruit of diastolic tension. The statement is made that when heard very close to the systolic, the diastolic gallop indicates concentric hypertrophy, and that the lengthening of the time between the abnormal diastolic and the first heart sound indicates a proportionate dilatation of the heart. The mesosystolic gallop is an index of weakened systole, and in general the diastolic is a sign of high, and the mesosystolic gallop a sign of low arterial and so endocardial tension.

Prognosis.—The most valuable contribution of the year comes from the pen of Sir Andrew Clark,[72] in a statement of clinical results from private practice. He presents 684 cases of cardiac valvular disease which had existed for not less than five years without causing serious symptoms. The primary and perhaps most important deduction from this list of cases is that a murmur *per se* has little or no influence in determining the prognosis in any given case. While medical opinion had been gradually tending in this direction, it had been helped along only by isolated cases. It is specially noteworthy that of this large number none came seeking relief from heart disease. By far the larger proportion, however, were suffering from conditions referable directly to the cardiac disturbance. Nearly one-half sought relief for gastric disturbance, and about one-fifth were suffering from nervous symptoms such as might be due to cardiac disturbance. As a result of Dr. Clark's paper, cases were reported in large numbers in which cardiac disease had been known to exist for variable periods of from a few years to twenty or thirty years. Among the more important and definite were some dozen cases by Dr. Gairdner.[73] Before considering Dr. Clark's conclusions, it seems fair to state that, although these 600 odd cases were obtained by exclusion from a still larger number, doubtless, who, having heart disease, did die, they fairly represent all cases of recovery from heart lesions; and occurring under his observation during a definite period, therefore represent those other physical conditions which favor prolonged life with, or recovery from, valvular disease. Basing his opinion both on facts and his own matured judgment, Dr. Clark concludes: (I) That many persons live an active business life and reach an advanced age without knowledge of an existing heart lesion which may have been present for years. (II) That the mitral regurgitant murmur following chorea usually disappears in eight or nine years. (III) That the effects of valvular inflammations do in some cases disappear, leaving no clinical evidence of their former existence, and that although this more frequently happens in young subjects, it may occur in older patients. (IV) That rarely the valvular defects resulting from degenerative processes in middle life disappear, and that when circulatory or respiratory disturbances accompany their commencement, they sometimes subside, and admit of apparently complete readjustment. The conditions which justify a favorable prognosis in any given case, according to Dr. Clark, are as follow:

(1) Good general health. (2) Just habits of living. (3) No exceptional liability to rheumatic or catarrhal affections. (4) Origin of the valvular lesion independently of degeneration. (5) Existence of the valvular lesion without change for over three years. (6) Sound ventricles of moderate frequency and general regularity of action. (7) Sound arteries with a normal amount of blood and tension in the smaller vessels. (8) Free course of blood through the cervical veins. (9) Freedom from pulmonary, hepatic and renal congestion. Most naturally, in making comparisons between different cases or in any given case, the location of the lesion becomes all important, and in this respect old established rules still hold good. The ratio of danger between aortic and mitral regurgitation would be extreme, and conditions which would justify a favorable prognosis in the latter would no more than offer slight hope of a long life in the former case. · In the discussion following Dr. Clark's paper, Dr. Clifford Allbutt laid special stress upon the habits of life, condemning unqualifiedly the use of alcohol, and less severely that of tea and tobacco. He considers the loud rasping murmurs, when of regurgitation, as of less importance than the "murmurist" one, and thinks the position of the apex, the signs of hypertrophy or dilatation, and the condition of the general circulation, are points of special importance in prognosis as regards the valve affected. Dr. Allbutt never accepts patients with aortic regurgitation for life insurance, but does often take cases of mitral disease under restrictions. Drs. Bristowe and Luch both reported cases substantiating Dr. Clark's conclusions, and emphasized the point that the intensity and character of a murmur are of comparatively little importance. Dr. Donaldson, of Baltimore, called attention to the fact that most of the 684 cases were systolic in rhythm, *i.e.*, aortic obstruction and mitral regurgitant, and so, from a mechanical point of view, the lesions most readily allowing of compensation. In many conditions disappearance of a murmur may be a bad rather than a favorable sign, as it may be due to dilatation of the valvular orifice and the formation of so large an opening as not to obstruct the regurgitant blood current. In this connection may be mentioned the report by Dr. Saudby[74] of four cases in which aortic regurgitant murmurs disappeared without apparent cause, and the valves were found incompetent at autopsy. In such cases the secondary cardiac changes must alone determine the

prognosis; and as they are usually in the nature of dilatation, the disappearance of a regurgitant murmur becomes a prognostic indication of serious import. The general consensus of opinion seems, then, to be in favor of a more favorable prognosis in a large number, if not all cases, of heart disease.

Treatment.—Much difference of opinion seems to exist in reference to both the general hygienic or constitutional, and the medicinal treatment of organic heart disease. All reporters agree, however, that a careful distinction should be made in cases of valvular lesions, between cases in which secondary compensatory changes have occurred and those in which they are not present or have become incomplete. Liebenstein[75] recommends Oertel's treatment in the earlier stages, when compensation is but slightly, if at all, deficient and in the first stage of fatty degeneration. It is to be used with great care and discretion. Nothnagel, on the contrary, opposes the Oertel treatment, except in conditions of degeneration. Dr. Franz[76] approves of the treatment in appropriate cases. For acute conditions he advises rest under all circumstances; but for chronic disease, even when so far advanced as to cause him to greatly fear the results, he has seen the heart's action become regular, stronger, and even palpitation decrease and all the subjective symptoms improve. He admits, however, that every case must be watched with great care, and that it is not safe to apply the same method to all cases indiscriminately. Schott, in discussing the question, stated that he had seen some very bad results from these cures of Oertel, and Nothnagel distinctly states that he considers this treatment entirely inadmissible in all cases where there is degeneration of the heart muscle. But he recommends most heartily gymnastics, under proper supervision, as of special value in dilatations, and available in nearly all forms of degeneration following valvular lesions. In connection with the exercise he also advises cold carbonated baths, claiming that their results are more lasting, though less quickly obtained than from exercise. Our own opinion is decidedly against Oertel's treatment in any but exceptional cases. It can hardly be claimed that the heart fails to compensate for all valvular lesions so long as full nutrition of the cardiac muscle is maintained. Oertel's treatment in valvular disease must act favorably by improving general nutrition, clearing the system of excrementitious products and furnishing abundant supply of oxygen. It certainly must act unfavorably by calling upon an already overworked heart for still greater exertion and bringing it nearer the point where force expenditure overbalances nutritive repair. Perhaps in a few cases the favorable may seem to exceed the unfavorable action, and the patient apparently improves. We believe, however, that it will occur only in cases where the general nutrition is affected by faulty action of the assimilative organs, and that even in such cases a thorough system of passive exercise will accomplish as much and often more than the Oertel treatment. Upon a basis of six cases treated by leeches over the liver, Dr. Shattuck,[77] of Boston, advises even more decided reduction of the amount of blood in the venous system by venesection in cases where the right heart has become overdistended and is failing in its function, whether the failure be due to overdistension from obstruction in the lung, to weakening of its walls through degenerative changes, or to a combination of both. Huchard[78] also advises blood-letting, when the circulation is failing through serous infiltration, to be followed by digitalis. Barr[79] advocates a withdrawal of blood to relieve the right heart, and suggests aspiration of liver or opening of hæmorrhoidal veins. His claim that the right heart can not be relieved by venesection of the arms "because blood cannot be drawn backwards in the veins," appears unfounded, as it is required simply to lessen the amount passing to the heart that its congestion may be relieved.

Among the many new drugs recommended for use in the various valvular diseases and for the relief of the consequent degenerations, none seem to have fully displaced digitalis. Indeed, the question at present is not so much which shall take the place of digitalis as in what conditions they may become adjuvants; strophanthus claims the first consideration for it, if any drug may lay claim to greater virtues than digitalis. Dr. Frazer's conclusions seem to have been borne out by the observations of most reporters. They are: (1) the action of strophanthus is more rapid and durable than that of digitalis; (2) it has no cumulative effects; (3) it produces no gastro-intestinal trouble; (4) it dilates rather than contracts the arteries, and so assists rather than obstructs the heart's action. From Dr. Haas'[80] experiments it would seem as though its action on the vessels in causing their dilatation was perhaps its most valuable element. He claims that the visible apex beat disappears, that it is weakened and diffused under touch, and that though the pulmonary second sound loses its accentuation, that the frequency of the pulse is diminished and the characters of the graphic record are markedly affected. It seems, as he expresses it, to cause such paralysis of the vessels that they appear to drink up the blood without arresting the circulation. He also thinks that it acts more surely and quickly than digitalis. In experiments on dogs, Lépine[81] found that a lethal dose stops the heart in diastole with a precedent increase in arterial tension. In the frog it increased the diastole when applied directly to the heart. Dr. Hochhouse[82] states, from an experience in 60 cases, that in the non-compensatory stage of valvular disease strophanthus is often a remarkable remedy, slowing the pulse, regulating its rhythm and giving it a firm, reliable character, though it failed to accomplish expected and desired results more frequently than does digitalis. In more chronic conditions of degeneration, he found that a rapid and irregular pulse was slowed and rendered even and regular, the dyspnœa was promptly relieved and the regulating power of the drug was specially manifest in this class of cases. All reports seem to agree that the special value in strophanthus arises from the fact of its action on the heart not being associated with increase but rather decrease of arterial tension. In this particular it seems to have a special advantage over digitalis, which perhaps no combination of an arterial sedative with digitalis could equal. Hill and Hutchinson,[83] in a report of cases, lay special stress upon this difference from the action of digitalis.

Many reports have been made based upon one or more cases and a more or less extended experience with the drug, and while they all support to a greater or less degree Dr. Frazer's original conclusions, there is as yet no evidence to show that strophanthus can supersede digitalis, but that it will be a valuable adjuvant and a substitute in those cases where digitalis is not tolerated, or where, from constant use, it has become less effective. In early conditions of cardiac weakness, the decrease in arterial tension may prove a sufficient relief to the heart for a time.

Glonoin, nitro-glycerine, is claiming much attention as of value in valvular or organic disease of the heart. Its action upon the heart is secondary, however, rather than primary, as it increases the capillary circulation and thus relieves the overworked heart. It seems of special value in all stenotic conditions, though there appears to be no reason why it should not be of service in any form of valvular lesion or degeneration of the myocardium. If in aortic stenosis it decreases the obstruction to the blood current, and so assists the ventricle equally in aortic regurgitation, it must render the aortic recoil more efficient in propelling the blood forward and lessen the amount which is thrown back into the ventricle. Barr[79] claims that there is no drug to be compared with glonoin in mitral stenosis, saying that it tends to deplete the pulmonary circulation. We are unable to see why it can act with special force upon mitral disease. Von Holst[84] further considers glonoin as a cardiac stimulant and nervine, and finds it of special value in conditions of cardiac feebleness attended by little or no degenerative changes. Bartholow[85] arranges its physiological actions under three heads:—(1) Lowering of arterial tension with slowing (?) of circulation. (2) Increase in the heart's movements. (3) Decrease of nervous irritability. Upon this physiological basis he advocates its use in all conditions where high arterial tension or weakening and degeneration of the heart muscle are reacting unfavorably upon the circulation. Secondarily, it acts to relieve the right heart congestion, but it does not have any such stimulant or tonic action upon the heart muscle as do digitalis, strophanthus, etc. In the earlier stages of valvular disease it may lessen the work done by the heart, and so postpone cardiac failure; and in the later conditions where failure is due to either secondary or primary degeneration of the heart muscle, it may alone be sufficient for a time and is usually a valuable adjuvant to digitalis.

Several clinicians advocate the use of adonis vernalis, and Herrman,[86] of St. Louis, considers it fully equal, if not superior, to digitalis. Seifert,[87] of Wurzberg, Leifert and others recommend caffeine as also a powerful cardiac stimulant. It has the advantages of rapid action and no cumulative effects. Very few reporters take occasion to instance the conditions in which various cardiac tonics are to be preferred, but Nothnagel's[83] general statements may be accepted as indicating the generally relative value of these drugs. In a recent lecture he states that he still prefers digitalis to the more recently advised remedies, and that he does not prescribe convallaria unless digitalis has failed. Adonis vernalis he uses in doses of 60–75–90 minims of the tincture in 24 hours. Caffeine is preferred to sparteine in cases where the rhythm is specially disordered, and is given in repeated doses of 3–5 grains, increasing to 30–40 grains per day. Nothnagel uses the double salts by choice. For anasarca he uses calomel and opium.

A more exact classification of the conditions demanding treatment is made by Huchard.[78] He recognizes four conditions of cardiac systole: (1) Ensystole, in which compensation is perfect and good hygiene is alone demanded. (2) Hypersystole, when compensation is exaggerated and cardiac sedatives are required. (3) Hyposystole, indicative of asthenia of the myocardium and vessels. Failure of compensation may be slight or result in œdema and anarsarca. It is best treated by digitalis. (4) Asystole, in which degeneration is too profound to respond to digitalis. In such conditions caffeine is effective.

After all has been said in favor of new drugs, we are unable to see that the position of digitalis has been specially affected.

Jendrassik,[89] Biro,[90] Stitler,[91] Mendelsohn[92] and others speak of the use of calomel as a diuretic in cardiac disease with dropsy, but do not agree as to the conditions in which it is most valuable. We do not hesitate to say that it will be in cases associated with parenchymatous nephritis. Dryordin[93] prefers infusion of digitalis, convallaria or caffeine, for cardiac dropsy and places milk next these drugs in value. Beaumetz[93] also speaks to oppose tapping for cardiac ascites unless asphyxia be imminent. Among the more unusual means employed may be mentioned the hot external applications of Schott[94] at 140° F., the chloride and chalybeate baths which Schloz[95] considers the most powerful of all heart tonics, and the mud baths. Regarding the last, Dr. De Sandfort[96] states from several years' experience, that they lower arterial tension without quickening the heart's action, and thus afford valuable rest, particularly to rheumatic hearts. The direct action of the bath is, of course, intended for the rheumatic condition, its action over the heart being secondary. Keirnan[97] considers hellebore as a valuable cardiac tonic, especially in patients suffering also from mental disease. In Jaccoud's clinic,[98] in a case of scarlatinal valvular disease with mitral stenosis, aortic stenosis and insufficiency and consequent dilatation, a "régime of inanition with iodides and bromides" is directed to save the patient from fatty degeneration likely to supervene. We are unable to appreciate the *rationale* of such treatment. Finally, as to the use of digitalis, Schott[94] states that it is most valuable in myocardial disease with weakness, dilatation and degeneration, while Neuzold[99] says that in nearly every case in which digitalis has failed, there will be found fatty degeneration or other myocardial change. Clinically we have found that digitalis fails to increase heart power whenever any forms of degeneration of the heart-walls allows of extensive dilatations of its cavities, and in all cases of well-marked fatty degeneration of the heart.

ANGINA PECTORIS.

Etiology.—Duroziez[100] recently dwelt at length, before the Paris Society of Medicine, on the complete way in which Reeder, in 1821, anticipated most later conclusions, and especially credits him with distinguishing between true and false anginas. Reeder's enumeration of the chief causes is as follows:—

(I) Ossification or some other lesion of the coronary arteries, greatly diminishing their calibre, or such ossification of the aorta as diminishes the diameter of the aortic orifices. (II) The dilatation of the heart that accompanies the morbid states represented by ossified valves and stenoses at the aortic and pulmonary orifices. (III) Aneurism and ossification of the thoracic aorta. (IV) Lesions of chylopoetic organs, especially those of stomach, causing indigestions.

Liégeois, Gélineau and Gauthier have very fully endorsed each other's conclusions, but are recognized as protesting against some conclusions of Huchard.[101] Liégeois,[102] describing the causes of angina as predisposing and causative, thus classes the former: (1) organic; (2) nervous; (3) diathetic; (4) toxic. Of *the organic causes* he states that three-fourths of all cases may be attributed to atheroma or sclerosis of the coronaries or aorta, either one or both; of the others in order of frequency, he gives, as the only ones worthy of consideration, dilatation of gastro-hepatic origin, acute pericarditis, chronic periaortic pericarditis and cardiac symphysis. *Nervous causes* are enumerated as neurasthenia, spinal irritation, hysteria, Basedow's disease and mental affections. Again, he considers these as either neuralgic in type or pure neuroses. *The diatheses* may manifest themselves in the form of atheroma or arterial sclerosis, yet do not need any such intermediary. A gouty or rheumatic subject may have an angina as he might a neuralgia; the diabetic also. Metastasis, too, may occur, when in a gouty or rheumatic subject there is a decline of the articular symptoms, and may then involve the cardiac plexus. Among the *toxic influences*, tobacco, and especially cigarette smoking, are very prominent, especially when combined with bad hygiene and malnutrition. Much the same may be said of tea drinking as soon as neurasthenia is well established, and ergot and malaria are credited with one case each.

Ischæmia of the myocardium is the preparatory condition of angina. There may be no coronary stenosis or obstruction; simple endoarteritis or periarteritis may so impair the contractility of the arterioles that sufficient blood cannot enter the parenchyma of the heart. The consequent anæmia may give all the phenomena of angina in the same way that arterial obstruction elsewhere causes the frightful pains of senile gangrene.

A similar anæmia may be produced functionally in neuropathic anginas. It is the same vasomotor action in the coronaries that produces the accompanying phenomena of pallor, cold extremities and vertigo preceding the attacks. Pure anginal neuroses uncomplicated by coronary lesions are rare; but in those cases of sudden suppression of gouty and rheumatic pains, it is not unreasonable to suppose that hyperæmia of the cardiac plexus may be one manifestation of the metastasis.

The direct exciting causes named by Liégeois are: Any active efforts or movements, such as walking fast against the wind, emotions of love, anger or joy, reflex influences from errors of diet, menstruation, chills, pains, etc.

Gélineau,[103] who is known as the historian of the "epidemic" of angina on a French man-of-war in the year 1858, as well as the author of the most considerable treatise on angina, adopts many of Liégeois' statements, but gives prominence in his etiology to degenerations and lesions of the myocardium and of the pericardium and to intra-abdominal and intra-thoracic tumors. He dwells on syphilis and gout as important factors in that they may act either as diatheses or by causing arterial sclerosis.

Huchard[104] supports his affirmation that sclerosis is the characteristic pathological feature of the disease, on the evidence of seventy-five cases, and states that the non-recognition of this truth has led to treatment of the symptom, pain, instead of the disease. He explains the case[105] in which the coronary orifices were not obliterated by assuming that at some part of their course the arteries were not patent. When obstruction or obliteration of the coronaries occurs without angina, as often happens in the aged, it is assumed to have occurred so slowly that collateral circulation could be established. In other cases[106] the absence of the lesions, in spite of typical anginal developments, is accounted for by the assumption of a reflex spasm and contraction of the arteries; such he asserts to be the case when angina is induced by tobacco smoking.

Other recent clinical experiences illustrate various aspects of anginal manifestations. Leyden[107] reports four cases in which he attributed the angina-like paroxysms to neuralgia affecting the vagi nerves. In these the disease was associated with tabes dorsalis appearing late. Vulpian has recorded a case in which the cardiac symptoms appeared early. The evidence of such cases suggests a degenerative peripheral neuritis affecting the vagus.

Dr. Tilley,[108] of Chicago, gives the history of an interesting case in which anginal paroxysms, moderate in severity, were followed by great œdema and sudden death. The autopsy showed one of the coronary arteries almost entirely occluded, and just below the manifest atheroma an aneurism about the size of a large walnut had developed. The ischæmia had led to great thinness of the cardiac wall. Ewald[109] gives a record of death in collapse of an elderly woman with the symptoms of mitral stenosis. The autopsy revealed complete ossification of the coronary arteries. The patient had had no anginal symptom, only extreme dyspnœa.

A suggestive case is given by Mollière,[110] in which a paroxysm the of "angina" killed the patient. The autopsy showed behind stomach and above the pancreas a mass of degenerated lymphatic glands that compressed and destroyed the two cords of the great sympathetic, enveloping in their mass most of the elements of het solar plexus, in which were the terminal fibres of the pneumogastric.

Dr. Letulle,[111] of Paris, records the case of an elderly man, over 70, whose previous history had been one of perfect health, and who died with his coronaries completely obliterated by atheroma after two attacks of cardiac asystole. The autopsy showed amyloid degeneration throughout the myocardium. There had been no syphilis nor rheumatism. The pulsations were hurried and irregular and there was much œdema of the lower part of the body. Wille[112] reports two cases of characteristic angina that showed ossification of the coronary arteries. Potain[14] insists on the distinctly typical forms of angina due to tobacco and to gastric troubles, but admits that they are not fatal unless accompanied by fatty degeneration or some other lesion. He remarks that the tobacco habit does not bring tolerance with it, as is the case with most other poisons.

Westbrook,[114] of Brooklyn, reports a case with very rapid pulse, in which præcordial pain was radiated into the neck, down to left arm and into the left leg. It has returned twice at intervals of three years. He remarked the occurrence of a mitral systolic murmur during the paroxysm, and claims that it is explained by vaso-motor obstruction, causing temporary functional incompetence of the mitral valve through disturbance of the muscular power of the left ventricle. Huchard's[115] clinical experience has led him to rely on the following summary of results to distinguish between true and false angina:—

False angina is peculiar to women of any age, from gastric and arthritic trouble. It frequently recurs, lasts long, and the pain is at the lower or middle part of the cardiac region. *True angina* is most frequent in men of somewhat advanced age. It is seldom periodic, never spontaneous, and is usually caused by some effort or emotion attended by cardiac excitement.

In his contribution of November, Huchard refers to his former classification of the three distinct anatomical forms of cardiac arterial sclerosis, which are as follow: (1) A dystrophic cardiac sclerosis due to an endarteritis obliterans of the coronaries; this is consequent upon a regional ischæmia and the lesions of the myocardium take place as far as possible from the vascular lesion. This form is essentially ischæmic, has no inflammatory character, and should not be called a myocarditis at all. (2) A peri-arterial inflammatory cardiac sclerosis. (3) The mixed form combining both the former.

In the present state of medical science it is difficult to attach a special symptomatology to each of the first two forms of cardiac sclerosis. The ischæmic form, however, is the graver, and predisposes more to painful accidents, to angina, dilatation of the heart and asystole. But cardiac arterial sclerosis representing arterial cardiopathies can be sharply distinguished from the valvular cardiopathies. The former have their location in the heart and their origin in the arteries, and are characterized throughout the larger part of their evolution by great arterial tension, due to vascular spasm, either temporary or permanent. The hypertension which precedes and produces the development of arterial sclerosis is to be recognized by the *diastolic* echo of the aorta, which is totally distinct from Traube's metallic echo and from the tympanic bruit of Mussy, as also from the diastolic echo of the pulmonary artery, which is the index of exaggerated tension in the lesser circulation. The existence and the frequency of the arterial spasm is demonstrated, even at the outset of general arterial sclerosis, by paroxysms of syncope, partial chills, dyspnœa, an arhythmic heart, tachycardia or acute dilatation. Cardiac arterial sclerosis manifests itself in five principal forms: The pulmonary, the painful forms, the arhythmic, the tachycardiac and the asystolic. In the pulmonary we get the false or cardiac asthma. The *painful* form corresponds to angina pectoris, or simply to retrosternal or epigastric oppression or anginal pseudo-gastralgia. The *arhythmic* form is very frequent and important. There may be an occasional manifestation of arhythmic action, or it may be permanent until a sudden asystole supervenes. Disturbance in rhythm may be due to tobacco, dyspepsia, etc.; but when accompanied by hypertension it must be considered as symptomatic of cardiac arterial sclerosis. Of the *tachycardiac* form, Huchard remarks that he has often observed it in arterial sclerosis as a permanent or temporary weakening of the pulse on the left side. The *asystolic* form is the finale of nearly all the arterial cardiopathies. Its evolution is sudden and rapid and often takes the form of an acute dilatation; the arterial cardiopathies may even set out with asystole ending where the valvular lesions begin. It may also be brought on by the slightest causes, a simple bronchitis, emotion or digestive troubles, that act quickly on a myocardium which sclerosis has made ready for dilatation. Cardiac arterial sclerosis, he says, is latent and insidious in its evolution and outset, paroxysmal in its course, sudden and bruital in its asystolic explosion.

According to Huchard,[116] the causes of sclerosis of the arteries of the heart may be divided into three groups: (1) Toxæmia by alcohol, tobacco, malaria and lead; (2) the diathetic states of rheumatism, gout and syphilis; (3) physical, moral and intellectual overpressure. The sclerosed heart may be either hypertrophied or dilated,—generally it is both. Modifications of form and size are the direct consequences of the initial vascular lesion and not of the ensuing sclerosis. The common lesion is obliterating arteritis of the small coronary vessels, and it takes place slowly, so as to allow time for compensating hypertrophy and for sclerotic lesions to develop. If the arteritis is of more rapid progress, dilatation and alterations of the heart muscle fibres are the preponderant changes.

Symptoms.—Liégeois[117] states that the atheromatous form of the disease, as distinguished from the neuralgic, is characterized by an intense sharp pain, usually appearing after some exertion, located beneath or behind the sternum and radiating to the neck and left shoulder and arm, which is associated with the evidences of cardiac hypertrophy; sharp ringing or dry parchment second sound over the aortic valves, and hard radials. Such cases have usually suffered from dyspnœa on slight exertion, for some time. Auscultation may show an aortic murmur, or only a doubling of the sound in place of the sharp second sound. Such a diagnosis is far from absolute, however. Huchard[118] records a case of complete closure of the coronaries in which pain was never more than a sense of constriction at the elbow and wrist.

Treatment.—Liégeois[119] makes four different diseases to be treated, according as the angina is an organic or functional ischæmia, a hyperæmia of the cardiac plexus, or a simple neuralgia of that plexus. Whether ischæmia is the outcome of organic or functional conditions, the indication is to get back into the cardiac parenchyma the blood that it lacks. Nitrite of amyl fulfills this indication in two ways; it dilates the vessels and it stimulates the heart. The action of morphia is similar but much slower. All vaso-constrictors are capable of producing collapse.

Huchard's treatment for prevention and cure of angina with sclerosis is endorsed by Liégeois. It is to take up to three grains daily of iodides for fifteen days, followed by from two to twelve drops of the 1 per cent. solution of nitro-glycerine for the same period, continuing the alternations for one, two or three years.

He believes that the effect of the iodides is to diminish arterial tension, to stimulate the interstitial circulation of the heart, and to induce resolution of arterial fibrosis when not too far advanced. The advocates of this treatment affirm that the ratio of deaths in cases of organic angina has fallen from nine in ten, to from three to four in ten, and claims to base the statement on absolute fact. In functional angina, where there is the same ischæmia, Liégeois prescribes the same treatment as regards the amyl nitrite, but uses the iodides only for tobacco cases, in which the sclerosis is assumed to be latent. For the angina that consists in hyperæmia of the cardiac plexus, the French treatment prescribes counter-irritants; in cases of acute determination of rheumatism or gout, both at the joints and over the præcardial region, with salicylate of soda internally, tempering its depressing effect by atropine or morphia injections. For the anginal neuroses active counter-irritation, hypodermatic injections of morphia, and the long-continued course of iodides. Purely neuralgic angina will yield to morphia.

Gélineau[120] pronounces angina to be a distinctly curable disease; he sanctions much of Huchard's practice, but disagrees with his distinction of false and true angina, based on their relative curability. Huchard[121] himself prohibits the administration of digitalis, ergot, belladonna, sparteine, and whatever tends like these to increase arterial tension in angina, as well as antipyrine and cocaine, that are apt to produce syncope. Germain Sée,[122] on the contrary, advocates the use of antipyrine for angina and for relief of pain in aortic insufficiency and most other cardiac affections.

Huchard also affirms that the potassium salts have a toxic effect when taken continuously, for which reason he prescribes sodium iodide. Guyot[123] and Potain are among those who oppose Huchard's conclusions with an incredulity founded on their own experience of the iodide treatment, and on the fact that angina often disappears spontaneously,—this in spite of Huchard's forty cures and of the seven years' test which he claims as the basis of his results. Holst,[124] of St. Petersburg, testifies that nitro-glycerine used in angina has greatly benefited and sometimes cured serious cases. Prof. Laschkevitch,[125] of Kharkov, administered cocaine in ⅓ gr. doses and claims to have obtained relief of the symptoms within three days. He recommends that this treatment should be supplemented by the inhalation of oxygen.

FUNCTIONAL DISEASES.

Etiology and Pathology.—Mills,[126] of Canada, to whose work slight reference has been made in valvular diseases, offers some conclusions, based upon an extended series of experiments upon the hearts of the lower vertebrates, in reference to the physiological action of the cardiac nerves. His primary and chief conclusion seems to be that nervous force acts primarily and constantly as a neuro-trophic influence, through which functional power becomes stored within the muscle cell, rather than as an immediate stimulant during functional activity. Upon this basis he explains the continuance of gland-secretion, etc., after section of its nerve supply, and concludes that all nutrition, as well as function, depends directly upon a similar nerve influence. Beyond the usual supply, he also recognizes a residual nerve force, expenditure of which entails marked depression and finally collapse. Diminished nerve power is thus manifested first in the digestive and other organs to which such force is indispensable. The heart also suffers and may manifest its depleted condition by irritability, irregularity, or even acute dilatation. Such an explanation does not preclude the possibility of "molecular" changes which cannot be recognized under the microscope. Mills has formulated the law that the beneficent action of the vagus is in direct ratio to the needs of the heart at the time, and analyzes that beneficent action, calling the vagus a sympathetic that inhibits, and the sympathetic a vagus without inhibitory fibres, by the explanation that the constructive metabolism which is the effect of the vagus may balance the destructive metabolism of the sympathetic. The heart-muscle has the choice, so to speak, of being stretched or not, and does not elect the former as long as the balance of nerve force is maintained.

His attempt to solve the problem of the causation of the heart-beat runs thus: First, we have the modified amœboids of every cell and the contractility natural to protoplasm, the concatenation between cells and between the different sections of the heart, and the fact that pulsation lingers longest in that section of the heart where the primitive heart-beat began. Intra-cardiac pressure and the condition of nutrition, as determined by the blood and the nervous supply, are the other factors.

Pramberger[127] emphasizes that all troubles of malnutrition have a relatively long stage during which they are latent. In his study of cardiac weakness he has chiefly concerned himself about the results of insufficient oxygenation, leading to that particular "splitting up" which produces fat. Tracing the process further, he believes it to be well established that, in the chlorotic, the restitution of inogen is hindered by the same lack of oxygen in the blood, which must react naturally upon the ganglion cells of the heart, accounting for the transitory heart troubles of the chlorotic, and the more so in that we must attribute a quasi nerve function to the cells themselves of the myocardium, in virtue of which they perform an important part in the phases of systolic and diastolic action.

Rosenbach[128] designates neurasthenia vasomotoria the neurosis of the heart induced in the young by exhaustion from any cause, tobacco, alcohol, etc. He describes two stages: (1) The stage of excitement, marked by congestions of the face and hands, hyperæsthesia of the cardiac region indicated by palpitations, anorexia, insomnia, frequent micturition, etc. (2) The stage of depression, with great pallor and lassitude, palpitations persisting in spite of low heart power. It may be cured, he says, but often results in muscle change.

Richardson[129] distinguishes and very definitely describes six varieties of palpitations, viz.: (1) Simple, caused by nervous strain; (2) palpitation with occasional intermittency; (3) palpitation with permanent intermittency; (4) palpitation with organic cardiac disease; (5) extra-cardiac or arterial palpitation, which he refers to the motion of arteries of the cardiac axis; (6) muscular palpitation connected with the muscular columns of the diaphragm. He regards its origin as nervous, and thinks it is probably seated in the sympathetic chain. Such subjects are exceptionally sensitive in childhood and remain so.

Schnell[130] has collected and classed all observations relating to functional troubles under the head of mechanical cardio-vascular disorders produced by pathological conditions of the nervous system. He found in six cases of general progressive paresis six organic cardiac lesions. Duchenne, Hallopeau, Raynaud, Duval, Raymond and Déjerine have noted functional troubles in cases of bulbar paralysis. Ollivier and Halberton attributed dilatations of the heart to compression of the bulb, and Semmola to a paralytic cardiac ataxia. Fifteen cases of sclerosis gave three organic lesions which might have been attributed to typhoid fever and alcoholism. Schnell concludes that the cardiopathies associated with locomotor ataxia, which are supposed to be a common cause of heart trouble, were coincidences merely. His general conclusion in regard to the proposition that the sequence of material or dynamic troubles in the nerve cells, accompanied by palpitations, and lesions of malnutrition in the myocardium, with hypertrophy or dilatation, is that it can be verified only as regards the first two changes. Of peripheral nervous disorders, he claims that neuralgias of the left upper extremity can induce nutritive lesions of the myocardium. His physiological investigations confirm the observation that section of the vagi causes fatty degeneration of the myocardium; and he admits the possibility that it may be due to the abnormally active movements of the heart, and offers an analogy to the myocarditis of the overworked heart.

Wyss,[131] of Zurich, had at his clinic an interesting case of paroxysms of tachycardia in a peasant brought on by overexertion. The pulse went as high as 270, and palpitations were apt to occur with any attempt at work. Digitalis was the only remedy that gave any relief, quinine, amyl nitrite and morphia being powerless, the last increasing the trouble. Graham[132] and Bristow[133] mention similar cases, one terminating fatally. Keating and Edwards[134] talk of the vagi and sympathetics being perfectly phlegmatic or callous in cases of irritable heart, and say that a peculiar flushing or lividity of the cutaneous surface is sometimes noted. Le Clerc[135] declares that hysterical angina is to be diagnosticated by the aura and irregularity of the pulse started by psychical impressions. Functional cardiac disease has been found with great frequency among soldiers, both in its distinctly functional form and in those resembling organic disease. Duponchel[136] adds to Potain's three sites of inorganic murmurs, a fourth at the third left intercostal space, which he considers the most important of all, stating also that the murmur is mesosystolic. While such conditions are spoken of as functional, it is recognized that actual changes of tissue may be present. Carrieu[136] believes that the changes of overwork are produced by the obstruction due to congestion of the right heart and venous circulation, or from deficient oxygenation, one or both. Kelsh,[137] on the other hand, after clinical and post-mortem studies, lays special stress upon overactivity due to a special demand of all parts of the body for increased blood supply. Seitz and Curschmann[137] attribute part of the results of overwork to an accumulation of carbonic and sacolactic acids. Revilliet[136] also describes "ponosis" as an infection by the products of denutrition, assuming either the acute or typhoid form, the former appearing in the sudden breakdown during long or forced marches; and Peter,[136] in a paper before the French Academy, describes a form of auto-infection caused by elements formed in the tissues independently of the action of micro-organisms.

Symptoms.—Grasset's[136] description of the gradual development of organic disease, from the first congestion of functional trouble, is in full accord with Duponchel's classification, in which he recognizes three stages:—

(1) Arhythmic pulsations, causing palpitation and intermittent and irregular pulse. (2) Congestion and hyperæmia leading to a myocarditis, with hypertrophy or dilatation attended by temporary murmurs. A condition which may undergo cure. (3) Well-established endocarditis and myocarditis with permanent murmurs and ending in asystole.

He also classes the palpitations which are so common a symptom of functional disease as, (1) purely nervous or idiopathic; (2) sympathetic, and expressive of disease elsewhere; and (3) symptomatic, in which the evidence of some organic lesion indicates thus in part the intimate relations often existing between functional and organic disease. He also states that the overworked heart may assume either of two forms, the transient or permanent, the former of which is indicated by ready exhaustion, dyspnœa, palpitations, increase of cardiac dullness, indicating dilatation rather than hypertrophy, and a systolic or presystolic murmur heard with maximum intensity near the apex. Œdema and albuminuria are not present. In the severe form the lassitude is extreme, with somnolency, loss of memory and general apathy. Pain in the præcordial region is quite constant, and œdema and albuminuria are usually present. It may even pass to the typhoid state with fever and small pulse. Such a condition is usually quite distinct from the irritable heart, with its quick, small and compressible pulse, that quickly slows in the recumbent position, its sharp but diffuse præcordial stroke, muffled first, and sharp, distinct second sound. Robinson,[138] of New York, affirms previous statements as to the effects of excessive mental and physical exertion upon both the heart's action and nutrition. He seems to lean rather to the organic side of the pathology, even stating his conviction that the dilatation which is sometimes found after prolonged exertion has been the cause of a suddenly appearing mitral or aortic murmur. He believes that the symptoms of intermittence in the pulse has no complete intermission in the cardiac systole, until after distinct cardiac degeneration has taken place, and that the neurosal form rapidly passes to the organic. He has also found albuminuria in such cases. Salger,[139] of Baltimore, also distinguishes between the purely neurosal irritable heart of early life and the depressed heart of later life, which he describes as closely similar to distinct cardiac degeneration presenting weak and frequent apex beat, low morning temperature, decreased quantity of urine of high specific gravity, neurasthenic symptoms and heavy dragging sensations in back and limbs. Depressed heart tends to fullness of the right heart and veins.

Potain[140] thus describes a typical case of cardiac trouble caused by dyspeptic paroxysms after each meal. The palpitations were accompanied by irregular rhythm and the heart's movements were sometimes accelerated, sometimes slowed. There was dilatation during the paroxysms of dyspnœa, and the apex removed outward. Palpation gave a presystolic "shock," auscultation a gallop rhythm. The pulse was weak, soft and compressible, generally rapid. There was distress and an anginal præcordial pain, the attacks lasting one or more hours. In such case the heart may become permanently dilated, and tricuspid insufficiency and ascites might be the conclusion.

Dehio[141] classified the phenomena of the nervous disorders as follows: Angina, nervous palpitation and tachycardia, the second manifestation being intermediate in character between the sensory and the motoric disturbance. His sphygmographic observations led to his distinguishing two groups representing varieties of palpitation. In the one there is more rapid palpitation and more rapid ventricular contraction,—a condition that can be produced by atropine. In the second, frequency of the heart's "shock" only is produced, while vascular tonus and arterial pressure are lowered. He attributes the first group to transient paresis of the medullary inhibitory centre, while in the second group the same paralysis extends to the vaso-motor centre also.

Diagnosis.—Rosenbach[128] relies for the diagnosis of a functional neurosis on the etiology and the absence of organic lesion; the continuity of the symptoms and absence of exacerbations, the absence of signs of congestion, and the fact that respiration is not much modified, though there is a subjective feeling of dyspnœa.

Prognosis.—Stiller[142] inclines to the opinion that cardiac neurosis, with the exception of Basedow's disease, seldom leads to organic changes, and only when the heart has been greatly overworked for a long period; further, that they are seldom fatal, except in the rare forms in which they can induce organic changes, if we except cases of shock. For purposes of prognosis he divides them into: (1) Idiopathic neurosis, which mostly depends on personal idiosyncrasies; (2) reflex, in which cases the prognosis depends on the primary trouble; (3) those dependent on a general neuropathy.

Treatment.—Widemann,[143] of Berlin, uses a six per cent. solution of common salt, of which he injects twenty to thirty grams subcutaneously and repeats with ten grams once or twice a day. He has found the effect on the heart, when it is weak from various causes, in every way satisfactory, and that he could restore the heart's action temporarily in the dying. Habershon[144] says that no heart tonic, neither digitalis, alcohol, ether nor ammonia, is so useful as strychnia, in cases in which both the heart muscle and its nerves are exhausted. He injects $\frac{1}{60}$ to $\frac{1}{30}$ gr. hypodermatically. Rosenbach does not recommend electricity and hydropathic cures, but moral, hygienic and dietetic treatment, with bromides, iron, quinine, ergotine and cocaine. Richardson[129] says that treatment of palpitations should be chiefly moral for the relief of apprehension, and hygienic, flesh brush, moderate exercise, etc., etc. Digitalis with nitric ether is sometimes serviceable. Beverley Robinson[138] depends on light diet frequently repeated, with ignatia, pepsin, and other remedies for indigestion; also washing out the stomach. Blisters and thermo-cautery were sometimes the only effective means he found of relieving those forms complicated with gastric troubles. He seldom did good with digitalis as a heart tonic, and often found caffeine or convallaria succeed when they were used hypodermatically. For temporary good effects he found coca the most potent drug.

ANEURISM.

Etiology and Pathology.—No more suggestive clinical contribution has been made to cardio-vascular pathology than the report of Dr. Neve,[145] our Corresponding Editor, of Bombay, already referred to under valvular diseases. He reports upon a class of people living at high altitudes, varying from 5000 to 15,000 feet, and often doing heavy work at altitudes as high as 13,000 feet; yet he says that among forty thousand patients he has met with but five cases of aneurism, and that four of these occurred in a single year of severe famine. Notwithstanding this remarkable showing, Dr. Neve states that syphilis is both widespread and severe in its manifestations in the class of people to which he refers. His suggestion that where the habits of the people are not toward sudden and intense labor, syphilis will not attack the arteries, hardly seems a sufficient explanation, especially as he mentions heavy porterage duty at high elevations as occupying a portion of the inhabitants among whom his observations were made. We confess ourselves unable to explain satisfactorily such freedom from arterial disease. A suggestion is offered under valvular disease. Dr. Neve should supplement this report by one upon the other accepted causes of arterio-fibrosis and atheroma, particularly gout and cirrhotic Bright's disease. In this connection, Dr. Gairdner's[146] statement that strain and concussion of the heart stand among the most important causes, is specially pertinent. Shrady[147] describes the aneurismal diathesis as a strong predisposing cause indicated by an overactive heart, short legs, long body and strong muscular development.

The possibility of aneurism occurring in children is noted by Edwards and Keating,[148] and in those rare cases in which they also suffer from atheroma. In a report[149] of fifty-five cases, only five were in patients under twenty, the ages in these five ranging from infancy to twenty years. That atheroma is not always present in young subjects is illustrated by a case reported by Prof. Pel,[150] of Amsterdam, in which endocarditis and subsequent embolism had caused aneurisms of the submesenteric and femoral arteries in a girl of twenty. In this connection he calls attention to the necessity of examining the heart whenever aneurism occurs without traumatic cause. That acute inflammatory changes, as well as chronic, may weaken an artery to the aneurismal point, is well shown by Vinay's[151] report of a case of aneurismal dilatation of the arteries of the cardiac pillars associated with myocarditis, in a case of acute yellow atrophy of the liver. Later pathological reports lay more stress than formerly upon a purely degenerative change of the middle coat as the only and essential lesion leading to aneurismal dilatation. Indeed, the clinical fact that aneurism of the large vessels is infrequent in connection with fibroid and atheromatous changes which lead to apoplexy, had been recognized, even while atheroma was looked upon as the primary lesion in aneurism.

Upon a basis of fourteen cases, Spillmann[152] names the basilar and sylvian arteries as the most common situation of cerebral aneurism. The orifice of communication with the lumen of the artery is usually small,—a fact which explains the survival of patients for some hours after rupture occurs, although the aneurismal sac is rarely lined with clots. Rendu,[153] of Paris, reports an unusual case in which old pericardial adhesions, apparently by traction, had induced a myocarditis followed by atrophy and absorption of the myocardium near the centre of the left ventricular wall, and caused union between the endocardium and pericardial sac, with aneurismal dilatation. The coronaries were sound.

Symptoms.—Clinical reports have offered little in the line of new symptoms, but have rather tended to point out the frequency with which aneurisms occur without any symptoms. Among others two cases are reported from the clinic of Lancereaux.[154] In a patient who had died of pulmonary tuberculosis, a large aneurism was found which had eroded two-thirds of the adjacent vertebræ without causing any symptoms. The second was an aneurism of the transverse arch,—a form in which symptoms are usually most marked. Constriction of the œsophagus, recognized only by catheterization, was the only symptom. Bord and Tellier[155] also report a case of aneurism at origin of abdominal aorta, which gave no physical symptoms, but in which loud inspiration, prolonged expiration, with dyspnœa and cyanosis, led to a diagnosis of emphysema and cardiac asystole. Such cases may serve to illustrate the extreme difficulty of diagnosis in some cases and the frequent absence of all the usual symptoms. (Delbeg.[156]) When the aneurismal sac is of considerable size and its walls elastic, we may obtain a characteristic symptom in the low amplitude and sharp hook in curve of femoral pulse, the aneurism acting something like the air chamber of a steam pump. The frequency with which single symptoms are present in aneurism becomes in itself a symptom. The anatomical relations are such in the thorax that an aneurism may readily affect but a single organ. Still further, the same form of pressure does not always give the same symptom. Pressure upon a primary bronchus may cause but little, if any dyspnœa, when suddenly slight enlargement of the tumor may extend the compression to the bifurcation of the trachea and rapidly induce peculiar suffocative sensations, with extreme difficulty in expectoration. Goodridge[157] speaks of this as often a special symptom. Coates[158] calls attention to the frequency with which hæmoptysis is due to rupture of small aneurism in phthisical cavities or even of large extra-pulmonary aneurism, and emphasizes the necessity for care in the diagnosis. Baumann[159] records a case in which repeated chills were accompanied by a temperature of 104. The aneurism was only the size of a walnut and was located on the transverse arch. Its cavity was filled with clots and polypoid vegetations. Baumann's only explanation of the temperature is the suggestion that there may have been absorption of septic matter or bacilli, as in endocarditis. The effect which any sudden lesion involving more or less shock may have in causing an "explosion" of symptoms dependent upon other pathological changes, is well shown by a case of aneurism reported by Girode.[160] The rupture of a small aneurism with the pericardium in a patient with atheroma and interstitial nephritis, although the amount of blood effused was only eight ounces, was marked by very slow pulse, temperature below normal, rigid contraction of left extremities, suppression of urine and coma. The explanation given was uræmia from sudden suppression and reflex nervous action.

A single case reported by Hare[161] shows that overelasticity of the aorta may possibly simulate a true aneurism. Such elasticity, however, as well as non-aneurismal dilatation, is usually more diffuse and will simulate aneurism but rarely.

Treatment.—The medicinal treatment, as gathered from all sources, may be quite fully summed up in the one word,—iodides. Balfour,[162] Jaccoud,[163] Tussell,[164] Dana[165] and many others report the usual excellent results from the use of potassium iodide. André,[166] of Toulouse, reports two cases practically cured by the iodide of sodium; the potassium salt, however, seems to be preferred. In reference to the amount of the iodide which should be given, Balfour[162] places quickening of the pulse as an indication of an overdose. His plan is to place the patient in bed for three to four days before any of the iodide is given, in order that the heart may settle down to a normal rate, and this is taken as a standard. He then begins with 10 grs. and gradually increases the dose, diminishing it at once on any increase in the pulse rate. When the proper and full dose is thus ascertained, it is continued steadily for from three to six months. He does not consider it necessary to starve the patient, believing the action of the iodide upon the blood to be quite sufficient. Jaccoud has combined the bromides with the iodides in some cases. The earliest manifestations of a beneficial effect are usually disappearance of pain and vertigo, and diminution of pulsation and of the size of the tumor; Later, the tumor may become distinctly firm, from disposition of laminæ of fibrin, and all pulsation cease. From Jaccoud's statement in one case that galvano-puncture was inadmissible on account of an aortic regurgitation, which might allow clots to be driven into the left ventricle, it might be inferred that he approved of the procedure in certain cases. It would seem, however, as though the danger was quite as great from clots in the general circulation; and Lépine[167] states that this treatment is far more dangerous than the introduction of foreign bodies, of which he considers the watch-spring or catgut as the best. Charmeil,[168] however, pronounces the dangers from galvano-puncture, of hæmorrhage, syncope, embolism, weakened sac and suppuration, to be slight, and although he had eight complete failures out of ten and no actual cure in his fifteen cases, still considers that the results warrant further trial. Barwell[169] reports what is called a "new treatment," which consists in a combination of electro-puncture and the introduction of coiled wires, and states that failure was due solely to the fact that the aneurism was double, only one portion being treated. Personally, we have little faith in either method and prefer the iodide treatment, combined possibly with ergot in some cases. Bouillaud[170] reports a cure by the use of aconite, which seems to be used largely by the French. Potain,[171] however, states that to be of any value it must be given in toxic doses. He has treated one case of abdominal aneurism, in which the only symptoms were deep burning pain and a murmur obtained without pressure by digitalis and the bromides. Alluding to the treatment by pressure above the tumor, he states that it has succeeded but is decidedly dangerous. Goodridge[172] proposes blood letting and atropine hypodermatically. It can hardly be intended as the sole measure.

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FEVERS.

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GENERAL PATHOLOGY OF FEVER.

MACALISTER[1] reviewed, in his Gulstonian Lectures on The Mechanism of Fever, the existing theories and presented the doctrine of the nervous origin of fever in a form somewhat different from any hitherto definitely stated. Fever of necessity implies (1) a disorder of the thermotaxic mechanism; (2) an excessive production of heat associated with excessive chemical changes in the tissues, the excessive production being more or less than that of a healthy person in full diet (perhaps oftener less than more), but more than that of a healthy person on fever diet; and (3) that the bodily temperature, depending on the balance between production and discharge, fluctuates as one or the other is in the ascendant, and is not of itself a true measure of either, or of the consumption of tissue that may be going on. Of the tissues whose oxidation is in general the immediate source of the body heat, the muscles must be regarded as the chief contributors. First, the muscles form something like one half the whole mass of the body; second, when the muscles are in use, the blood which leaves them by the veins contains more carbonic acid than the blood even of the right ventricle. The mean rate of oxidation in them is higher than that of the average of all the tissues, including the muscles themselves. Lastly, muscular exercise in which the metabolism, as evidenced by the increased excretion of carbonic acid, is markedly increased, and is accompanied by a large concomitant increase of heat production. The heat production in the muscles, the chief producers of fever, is probably carried on under the influence of a two-fold nervous mechanism; the one part exciting to thermogenesis, accompanied by destructive metabolism; the other staying thermogenesis and subserving constructive metabolism. The thermogenic tonus is the manifestation of the mutual balance between these two parts. The nature of the nervous mechanisms subserving heat loss, is paralleled by an analogous twofold character in the nervous mechanism subserving heat production.

There is a certain order of progression observed in the disturbances of the thermal relations of the body. First and most easily disturbed is the thermotaxic nervous mechanism. If that only is deranged then result strange risings and fallings of temperature, as the independent variations of production and loss are concurrent or the reverse; but there need be no fever. The balanced rhythm of anabolism and catabolism in the muscles is not disturbed; there is no excessive oxidation and no excessive inhibition. This may be called thermal ataxia.

The next degree of disturbance is that in which there is not only thermal ataxia, but also disorder of the parts of the nervous system subserving heat production: there is underaction of the anabolic nerves, with diminished construction and diminished absorption of energy; there is overaction of the catabolic nerves, with increased oxidation and thermogenesis. The net result is manifest wasting or "combustion" and generally high temperature. This is ordinary pyrexia, and the nervous disorder does not generally go further.

There is another stage, namely, when the mechanism of heat loss is also profoundly disordered, so that the rise of temperature from the additional excessive thermogenesis does not stimulate it (or does not stimulate it enough) to meet the requirements of the latter, and the temperature reaches an excessive or even fatal height. This is hyperpyrexia.

Thus the thermal nervous system has three parts,—the thermotaxic or adjusting, the thermogenic or producing, and the thermolytic or discharging mechanism. Disorder of the first implies irregularity of temperature only; disorder of the first and second implies in general heightened temperature and increased body heat,—that is ordinary fever; disorder of all three implies in general hyperpyrexia, dangerous increase of heat and steadily rising temperature. In the ascending scale of evolution we seem to rise from the thermolytic to the thermogenic and, then to the thermotaxic nervous system. Cold-blooded animals possess the mechanism that in mammals becomes thermolytic,—a nervous mechanism which controls the vessels and the breathing. The thermogenic system in them is ill-developed, and probably not well differentiated from the system of motor and inhibitory nerves that subserves locomotion. The frog in his normal condition hardly needs to generate heat in his muscles; but when the motor trunks are artificially stimulated heat can be produced in them. But his oxidative thermogenic mechanism is at best feeble and we cannot easily throw him into an enduring fever. In young mammals the thermogenic system is developed before the thermotaxic. The chief characteristic of an infant's temperature is its instability. As the child grows, the range of its power of regulation increases, its temperature becomes stable and the thermotaxic mechanism is evolved. Regarding the thermal mechanism as a functional and evolutional hierarchy, Macalister looks upon fever as a "dissolution," a progressive negative process, a relaxation of control from above downward.

What happens in recovery from a typhoid febrile attack may be considered as a test of this dissolution hypothesis. First the thermolytic mechanism is aroused to adequacy; there is a critical sweat or a relaxation of the vessels of the skin, and a gush of heat from the surface brings down the temperature with a run. But the thermogenic centres have not yet recovered, and the temperature will swing backward and forward for some days and an epicritical excretion of urea takes place. Thermogenesis becomes less and less excessive, and is counterbalanced by thermolysis; but thermotaxis is yet feeble, and though the temperature is down it is still far from stable. As convalescence proceeds the stability increases; and at length thermotaxis, the first of the triad to be disturbed and overthrown, is the last to be restored.

T. J. Maclagan,[2] in an elaborate series of articles upon pyrexia and hyperpyrexia, contends that these two conditions, which have been linked together clinically and pathologically, and looked upon as degrees of the same condition, allied both in nature and causation, are essentially different in causation. The only band between them is the occurrence in each of a temperature higher than normal. The parts which go to form the thermal apparatus are: (1) the tissues in which heat is formed; (2) the surface from which heat is eliminated; (3) a central controlling power in the brain (a thermic centre); 4 nerves connecting this with the heat-forming parts of the body; (5) nerves connecting it with the heat-eliminating surface. The harmonious working of these different parts of the thermal apparatus gives rise to the phenomena of thermogenesis: and the general result is a persistent temperature of 98.4° F. Interruption of this harmony causes the temperature to rise or fall. There are two theories of fever with which we have to deal: (1) the combustion theory, according to which the rise of temperature results from increased activity of the process by which heat is naturally formed in the tissues; (2) the neurotic theory, according to which the rise of temperature is due to impairment of that inhibitory force by which the heat-producing process is kept within normal non-febrile bounds. These two theories are not antagonistic. Each is complete in itself, and fulfills the requirements of certain facts of pathology independently of the other. Each has its own special sphere of application. The neurotic theory applies probably to all cases of hyperpyrexia, to all rises of temperature resulting from the non-inflammatory lesions of the nerve centres, and probably also to most forms of ephemeral fever. By the combustion theory are to be explained the febrile symptoms of the specific fevers, malarial fevers, rheumatic fevers, pneumonia and all febrile diseases, of which the poisons may reasonably be regarded as minute organisms whose morbific action is dependent on their organic development. Maclagan regards it as probable that the fever of all inflammations is also thus produced, not that all inflammations are caused by foreign organisms (though many probably are), but because excessive activity in the minute cellular elements of the tissues may have the same effect on their metabolism as the propagation by them of foreign organisms.

The arguments by which the author's views are sustained is elaborate and ingenious. Especially ingenious is that portion of the paper in which he seeks to account in large part for the excessive waste of albuminous tissues and the excessive consumption of water in fever by the appropriation of nitrogen and water on the part of the contagion in its growth and reproduction within the body.

Austin Flint[3] is disposed to take a somewhat more narrow view of the mechanism of fever. He regards the cause of pyrexia in essential fevers, for example, typhoid, as twofold: On the one hand an exaggeration of the chemical changes taking place in the organism, by which, within natural limits, animal heat is generated; on the other, a disturbance of the processes of equalization of the heat of the body, mainly by the action of the skin. He regards water produced *de novo* in the economy as a product of excretion. The formation of water in health is mainly connected with the process of calorification. In fever the production of water seems to be diminished. His views are embodied in the following propositions, some of which are to a certain extent, novel:—

(1) It is probable that the original cause of most, if not of all the essential fevers is a microörganism, different in character in different forms of fever.

This proposition is based upon bacteriological researches of recent date, especially with regard to typhoid fever.

(2) Defining fever as an abnormal elevation in the general temperature of the body, the pyrexia is due to the following modifications in the normal heat-producing processes:—

(*a*) Oxidation of certain constituents of the tissues, probably by reason of the presence of microörganisms in the blood, is exaggerated independently of increased muscular work and without being compensated by a corresponding increase in the appropriation of nutritive material. This increased waste of tissue is represented by the excess of carbonic acid and urea excreted.

(*b*) The part which the formation of water within the body plays in the production of heat is either suppressed or is greatly diminished in prominence, together with the equalizing action of cutaneous transpiration.

This proposition is based upon clinical facts, which show an increased excretion of carbonic acid and urea, and a diminished excretion of water in fevers, and upon experiments which show that muscular work, while it increases heat-production, increases the production of water.

(3) Fever produces abnormal consumption of fat, with parenchymatous degenerations, for the following reasons:—

(*a*) The fat is consumed because it feeds the pyrexia more readily than do the other tissues of the body, and its consumption is the most important source of carbonic acid.

(*b*) Parenchymatous degenerations of muscular tissue and of the solid organs occur, chiefly because the abnormal transformations of these parts, which result in an excess of urea, and which probably, also, contribute to the excess of carbonic acid, are not compensated by the appropriation of nutritive matters from the blood.

(*c*) It is well known that patients with unusual adipose or muscular development are likely to present a more intense pyrexia in fevers than are those whose adipose and muscular development is smaller.

Finally. *An essential fever is an excessive production of heat in the body, induced by a special morbific agent or agents, and due to excessive oxidation, with destruction of the tissues of the body, and either a suppression or a considerable diminution in the production of water.*

Suppression or great diminution of cutaneous transpiration in the essential fevers, while it contributes, in a measure, to the rise in temperature, is not itself a cause of fever.

H. C. Wood[4] summed up our present knowledge of fever and its antipyretic treatment in the following propositions:—

(1) Fever is a disturbance of calorification in which, through the nervous system, heat dissipation and heat production are both affected. If there be a fever which is produced by the direct action of a poison, independently of the nervous system, we have at present no proof of its existence.

(2) Heat production is regulated by a nervous apparatus, of which the knowledge is still imperfect. There is certainly an inhibitory centre which depresses or controls the production of heat. It probably does this by acting on the trophic cells of the gray matter of the spinal cord. It is also probable that there is a centre which, when excited, increases tissue change, but its existence has not been absolutely proven.

(3) Heat dissipation is regulated through the vaso-motor nerves, so that vaso-motor paralysis is followed by an enormous loss of animal heat, and, under unfavorable conditions, by death from cold.

(4) Drugs may lower bodily temperature in health and in fever by increasing heat dissipation.

(5) It is conceivable that there may be drugs which, instead of acting simply on heat dissipation through the vaso-motor system, act absolutely on heat production through the inhibitory nerve apparatus. A drug which acts upon the vaso-motor system, increasing heat dissipation, may be spoken of as a false antipyretic;

and a drug which absolutely influences the production of heat, may be spoken of for convenience as a true antipyretic.

Wood claims that we have at present no positive knowledge about the mode of action of any antipyretic drug, except perhaps quinine and antipyrine. We believe that aconite, veratrum viride, and drugs of that class are false antipyretics because we know that they cause vaso-motor paralysis, and it is therefore probable that they increase heat dissipation and lower temperature; but we do not know whether they have an inhibitory influence or not.

Collie[5] does not definitely accept any of the views of the nature of fever at present entertained,—leaving the matter *sub judice*, with the statement that "before any theories can be formed of the pathology of the febrile state a clearer knowledge of the exact conditions which regulate and maintain the temperature in health must be acquired." He regards the extremely sudden onset of some fevers, especially those of a malignant type, as strongly militating against the view that their cause is a multiplying germ: he sees in it rather the action of a paralyzing influence acting at once on the nervous centres.

Hobhouse[6] regards the blood as one of the great instruments for regulating the body temperature; not as being in itself a heat producing agent, save indirectly and to that small extent to which metabolic processes actually take place in it,—which modern physiology is daily limiting.

Hösel[7] insists upon the view that it is not the presence of micro-organisms in the blood, but the action of certain chemical substances resulting from the functional activity and multiplication of these organisms which constitutes the primary causes of fever. He formulates this view as follows:—"Fever is the result of intoxication of the nervous system by chemical poisons circulating in the blood; and in every disease attended by fever, this symptom should be ascribed to the factors which produce these poisons."

Hösel arranges all febrile diseases from the stand-point of etiology, into two groups:—

(1) Febrile diseases due to the presence of specific microbes.

(2) Febrile diseases occurring without the introduction of specific microbes.

I. *Febrile Diseases due to the Presence of Specific Microbes.* —We must first consider the manner in which micro-organisms, with their chemical products, can affect the body.

The primary effect of lower organisms which have found access to the body will be that of foreign bodies of any kind. [In what follows dead and living foreign bodies are separately considered.] They either remain or are at once gotten rid of. In the latter case they have little or no effect on the organism, and require in this connection no further consideration. If microbes remain in the body, there are two possibilities: they either find a favorable nidus and develop, or finding none they perish. We will consider the last condition first:—

(*a*) Dead microbes remain in the body. What happens? They are either after a time eliminated or they become encapsulated; that is, they act as dead and sterilized substances. Example: calcified tuberculous glands. The process is analogous to that by which a bullet becomes encapsulated or the encapsulation of trichinæ takes place.

(*b*) What takes place in other cases? Microbes remain in the body; find conditions favorable to their growth; undergo development, and produce chemical substances. They act as living foreign bodies. (1) At first only in the affected region or in the affected organs to which the microbes have gained access, are active chemical processes and the production of by-products possible. According to Virchow, the cells of the infected region tend to separate the intruders as foreign bodies. The body responds to the irritation with a local inflammation, of which the tendency is to cast out the microbes and their products. This may succeed, and microbes and ptomaines are directly gotten rid of. In this case we have to do with a local inflammatory process, but no fever. Example: local tuberculosis without fever.

(1) Pulmonary phthisis in later stage. Tubercle bacilli have become fixed in the tissues, have there excited inflammation, have produced chemical products, the nature of which is as yet unknown. These inflammatory and other products are, however, after breaking their way into a bronchus, voided in the sputum. (2) Local bone tuberculoses in later stage. After fistulæ have formed by which the microbes and their products find their way out of the body, there remains merely a local process: no fever.

(*c*) What happens when the ptomaines and their products can not be gotten rid of, and find their way into the blood?

A distinction must be made between the cases in which the microbes themselves find their way into the circulation, and those in which the chemical poisons alone reach it. In the first instance, that is, where the microbes also reach the blood, we pass by the cases in which there is mere metastasis, as simply repetitions of the original local process. Let us consider only the cases in which the microbes remain at the point of infection, but their chemical products find their way into the blood. What is the result? There is forthwith poisoning with these substances, and we have to deal with local process and fever. Example: acute infectious diseases: (1) croupous pneumonia: local processes in the lung produced by cocci; fever by their chemical products; (2) diphtheria: local process in the mucous membrane of the respiratory tract produced by bacteria, fever by their chemical products. [It may be remarked in this connection that the diphtheritic membrane is probably produced by the union of diphtheria ptomaines with some chemical tissue element of the mucosa.]

In febrile diseases, in the production of which specific microbes are concerned, the local manifestations are produced by the microbes, which act as living foreign bodies and are treated as such by the organism (inflammation), while the fever is the result of an intoxication of the nervous system brought about by the action of chemical products due to the growth and functional activity of the microbes.

II. *Febrile diseases occurring without the introduction of specific microbes.*

The fever is here to be explained in the same way as in the first principal group. The only difference is in the source of the toxic principles. There the chemical products of metamorphosis are due to microbes; here the body itself is the laboratory in which they are formed. There are ptomaines developed in the course of physiological processes to which by way of distinction Gautier has given the names of leucomaines. Chemical poisons of this kind, developed under certain conditions and finding their way into the blood, are capable of exciting the characteristic phenomena of fever. Example: constitutional diseases: anæmic fever; the fever of subcutaneous fractures, etc.

We may therefore attribute certain fevers arising in the absence of specific germs to similar causes, and regard these forms of fever likewise as due to intoxication of the nervous system by chemical poisons circulating in the blood.

The Separate Forms of Fever.—A distinction is to be made between the intensity and the course of a fever. The intensity must be measured by the height of the temperature, the heart's action, the degree of constitutional reaction in general, etc. The course or type of the fever may be continued, remittent, intermittent.

The intensity and duration of a fever depend upon,—

(1) The productive coefficient of the microbe or of the organism itself.

(2) The degree of poisonousness of the products of metamorphosis.

(3) The capacity of the organs of elimination.

(4) The oxydative capacity of the organism.

The productive coefficient means the amount of chemical product, which a given number of microbes or the organism itself may produce in a given time.

The above factors explain all forms of fevers.

(1) Continued fever. The productive coefficient remains constant; elimination and oxydation diminished.

(2) Remittent and intermittent fever. Intermittent production.

(3) Lysis. Production gradually decreases. Elimination results slowly.

(4) Crisis. Production ceases at once. Elimination follows promptly.

(5) Hyperpyrexia. Uninterrupted rapid and abundant production and absorption, elimination diminished, etc.

(6) Death. Uninterrupted production. Arrested elimination.

(*c*) Handfield Jones[8] collected an important series of cases illustrating the production of high body temperature in various anomalous conditions. He holds the view which he advocated ten years ago: "that while the actual generation of heat doubtless depends upon the combustion of protoplasm, the amount produced is regulated by the nervous system, or more exactly, by certain nervous centres probably located in or near the pons varolii."

Those who adopt this theory regard fever as a paralytic phenomenon. So markedly paretic are some of these disorders that accompany high temperature that Sir Thomas Watson wrote in his lectures the sentence, "Debility is an original and essential part of fevers." Jones thinks that fever motors affect the temperature centre in the same way as they affect the intellectual, muscular or any other centre; that is, they paralyze the nervous tissues to a greater or less extent. The assumption of the existence of a regulating mechanism for temperature is largely justified by a knowledge of a similar controlling mechanism in the case of the heart. Excessive rapidity of cardiac action is noticeably a paralytic phenomenon, just as pyrexia seems to be. He cites cases illustrating the instability of febrile temperature as compared with the stability of normal temperature under the influence of hot and cold applications, in confirmation of this view. Reflex or inhibitory paralysis and exhaustive or primary failure of nerve force are among the dangers of the thermic nervous mechanism and therefore demand consideration as among the factors of fever.

Sylva and Pescardo[9] observed during the febrile period of several diseases a marked increase of electrical excitability of the cubital nerve. The difference was greater by the constant than by the faradaic current. This condition subsides under the action of antipyretics and upon the decline of the fever. The same effect is obtained by the bromides, which, however, in hysterical subjects appear to increase the nervous excitability. Zinc oxide gives uncertain results. Camphor at first weakens, then increases the excitability. Chloral, after a momentory increase of excitability, lessens it, while morphine, injected either in the neighborhood of the nerve or elsewhere, lowers it markedly. Pain lowers it and massage of the forearm for ten minutes markedly lessens it for some time. The continued administrations of the above medicaments does not affect the daily oscillations of nerve-irritability.

Bokai,[10] as the result of an experimental study of the intestinal movements, reaches the following conclusions:—

(1) The constipation usually present in fever patients, which is also a result of lessened quantity of food taken, is better explained by the increased irritability of the inhibitory nerves of the intestine; (2) this increased irritability is dependent on and a result of the

rise of temperature; (3) when the rectal temperature in artificially heated animals is over 102.2° F., and below 108.5°, the increased irritability may be observed, and it ceases on injecting 0.04–0.05 gram of hydrochloride of morphia; (4) a temperature exceeding 108.5° leads to paresis of the inhibitory nerves.

Amiel[11] studied the much disputed question of the relation between the evolution of syphilis and the eruptive fevers. It now appears to be established that certain, at least, of the eruptive fevers arrest for a time the course of syphilis. The syphilitic manifestations, however, reappear after a time and run their course as before. Two modes of action are to be considered. First, a profound general modification of the organism under the influence of which the diathesis disappears; and second, an influence purely palliative, under which by a local revulsion stubborn atonic ulcerations or intractable eruptions vanish. Diday has recorded a case in which rebellious syphilitic manifestations underwent retroversion upon the appearance of an outbreak of boils. Many observers have studied the disappearance of syphilitic eruptions after erysipelas. It is difficult to say with precision what the influence of syphilis upon the eruptive fevers is. One positive conclusion has been reached, namely, that the eruptive fevers are as a rule more grave in debilitated subjects and those suffering from antecedent disease than in healthy individuals free from previous disease.

Nevin[12] held that the immunity of communities and of individuals from severe attacks of specific fevers is due to an adjustment of the organism to the causes of such diseases,—an adjustment of which the tolerance for tobacco gradually acquired by those who "learn to smoke" is an apt illustration. This adjustment or acquired tolerance is capable of hereditary transmission like some other acquired peculiarities. He sums up his observations in the following words:—

"In the self-protective fevers the immunity conferred by an attack is probably due to a change in the nature of the protoplasm of the tissues. This change is probably effected by the chemical products of the fungus causing the fever, or by the dead fungus. These chemical products are adequate to explain all the other phenomena of fevers, and afford as good an explanation as any we have of the rise of temperature.

"The immunity acquired is to some extent handed down from one generation to another, and tends ultimately to ameliorate the character of the self-protective fevers."

Zweifel[13] found that in various diseases of the stomach and in fevers the absorption by the gastric mucous membrane is retarded. His method of testing consisted in the administration of small amounts of potassium iodide 0.2 gram (grains iij) in capsules, followed by a draught of water, and testing for the appearance of the drug in the saliva, exceptionally in the urine. The presence of iodine is recognized by the red, then blue discoloration upon the application of a drop of hydrochloric acid to starch paper moistened with the saliva. In healthy persons the red color appeared on an average in eight and a half minutes, the blue in ten and a half minutes. The appearance of the drug in the urine was a little later. These observations related to the empty stomach. After the taking of food the absorption was much delayed. In fever the iodide is much more slowly absorbed than in health. As a rule the mere intensity of the fever has little or no influence upon the rapidity of absorption; yet it takes place more rapidly in a declining than in a rising temperature.

Nevin[14] alludes to the diagnostic significance of the special odors which attend some of the infectious diseases. The odor of small-pox he states occurs only in the worst cases, and he ascribes it to ptomaines generated in the process of tissue necrosis. He speaks of a peculiar, sweet, almost aromatic odor of the breath observed in scarlet-fever patients, most marked in the early stages of the disease. Somewhat similar odors, which he states may be readily recognized, are noticed in typhoid fever and measles.

THE TREATMENT OF FEVER.

General Considerations.—Collie[15] is a believer in the rational use of alcohol in the treatment of fevers. He says "whilst alcohol is sometimes a useful and sometimes an indispensable article in the treatment of acute diseases, it is not required in all cases." . . . "The quantity to be given in any particular case must vary widely. The age, sex, personal habits and history of the patient must be considered; his general condition as to strength or weakness, his appetite, his sleep or want of sleep, the condition of his mind and his idiosyncrasy as to alcohol. The nature of the disease will also have to be considered,—its severity, its period and its complication. There is no such thing as so much alcohol for a person or so much for a disease." He prefers[16] high class champagnes and burgundies or old port, and when these can not be obtained, Scotch whisky. He thinks that a fair quantity daily for each adult patient is one bottle of champagne, or burgundy, one-half bottle of old port, or six ounces of whisky or brandy. So highly does he value alcohol in infectious diseases that he gives it in preference to opiates and chloral to induce sleep.

Collie is by no means an advocate for the routine use of the cold bath in the treatment of fever. He says, in speaking of the means of procuring sleep: "A warm bath may be given, especially in the case of young children and elderly persons; but in the case of young adults, who are not very bad and who preserve their strength fairly, a cold bath of about ten minutes' duration, at a temperature of about 60° F. may be administered. But the cold bath should not, in the writer's opinion, be given in typhus, influenza, relapsing fever, diphtheria, erysipelas, malignant fever, meningitis, hæmorrhagic small-pox or measles; nor in cases where there is hæmorrhage from the bowels, peritonitis, cardiac weakness, pneumonia, nephritis, or strong dislike for the bath on the part of the patient;" and he adds, "In the case of hyperpyrexia, however persistent, from 105° F. and upward, whether in young or old, a bath more or less cold—regard being had to all the circumstances of the case—should not be omitted, except when the hyperpyrexia comes on quickly at the end of the disease and is the expression of rapidly approaching death."

Austin Flint[17] has been led by his reflections upon animal heat and fever to present certain views, summarized in the following propositions:—

(1) Fevers, especially those belonging to the class of acute diseases, are self-limited in their duration, and are due each one to a special cause,—a microörganism, the operation of which ceases after the lapse of a certain time.

(2) We are as yet unable to destroy directly the morbific organisms which give rise to continued fevers; and we must be content, for the present, to moderate their action and to sustain the powers of resistance of patients.

(3) The production of animal heat involves oxidation of parts

of the organism or of articles of food, represented in the formation and discharge of nitrogenized excrementitious matters, carbonic acid and water.

(4) As regards its relations to general nutrition and the production of animal heat, water found in the body by a process of oxidation is to be counted as an excrementitious principle.

(5) Fever, as observed in the so-called essential fevers, may be defined as a condition of excessive production of heat, involving defective nutrition or inanition, an excessive production and discharge of nitrogenized excrementitious matters and carbonic acid, with waste and degeneration of the tissues, and partial or complete suppression of the production and discharge of water.

(6) Aside from the influence of complications and accidents, the ataxic symptoms in fevers, the intensity and persistence of which endanger life, are secondary to the fever and are usually proportionate to the elevation of temperature. These symptoms are ameliorated by measures of treatment directed to a reduction of the general temperature of the body.

(7) The abstraction of heat by external cold and the reduction of temperature by antipyretics administered internally, without affecting the special cause of the fever, improve the symptoms which are secondary to the pyrexia.

(8) In health, during a period of inanition, the consumption of the tissues in the production of animal heat, is in a measure saved by an increased production and excretion of water.

(9) In fever, the effects of inanition, manifested by destruction and degeneration of tissues, are intensified by a deficient formation and excretion of water.

(10) Alimentation in fever, the object of which is to retard and repair the destruction and degeneration of tissues and organs, is difficult mainly on account of derangements of the digestive organs; and this difficulty is to be met by the administration of articles of food easily digested, or of articles in which the processes of digestion have been begun or are partly accomplished.

(11) In the introduction of the hydrocarbons, which are important factors in the production of animal heat, alcohol presents a form of hydrocarbon which is promptly oxidized, and in which absorption can take place without preparation by digestion.

(12) Precisely in so far as it is oxidized in the body, alcohol

furnishes matter which is consumed in the excessive production of heat in fever, and saves destruction and degeneration of tissue.

(13) The introduction of matters consumed in the production of heat in fever, diminishes rather than increases the intensity of the pyrexia.

(14) As the oxidation of alcohol necessarily involves the formation of water and limits the destruction of tissue, its action in fever tends to restore the normal processes of heat-production, in which the formation of water plays an important part.

(15) The great objects in the treatment of fever itself are to limit and reduce the pyrexia by direct and indirect means; to limit and repair destruction and degeneration of tissues and organs by alimentation; to provide matters for consumption in the abnormal production of heat; and thus to place the system in the most favorable condition for recuperation after the disease shall have run its course.

Albert Robin,[18] at a meeting of the Paris Biological Society, communicated further details of his researches on the subject of fevers. In typhoid fever, more especially, medicaments should be employed, which increase oxidation, such as oxygen, cold baths, cutaneous derivatives, chlorate of potash, the iodates and the bromates. Substances like sulphate of quinine and antipyrine, which diminish oxidation, should be avoided. Chlorate of potash, the iodates and the bromates, must be given very cautiously, owing to the poisonous effect of too large a dose. Robin thinks that they may be replaced by drugs which indirectly favor oxidation, such as alcohol, copious draughts of milk, etc. D'Arsonval stated that the results obtained by the use of cold baths as described by Robin, agreed with his own investigations on the temperature of animals. In animals, however, oxidation did not increase heat.

Dujardin-Beaumetz[19] sought to revive the use of alcohol in the treatment of fevers in accordance with the principles laid down by Dr. Todd. In advocating alcoholic stimulation in fevers, Dujardin-Beaumetz lays stress upon the results of physiological investigations which have led him to regard alcohol as a tonic, an aliment and an antipyretic. To these conclusions the profession at large to-day yields only a qualified assent. While a large majority still contends that alcohol in carefully-regulated amounts is a tonic, the number willing to regard it as a food in the true sense, is not so great; and finally those who admit it to be either a safe or certain antipyretic is very limited. Alcohol shows its antipyretic and its toxic effects simultaneously.

Glax[20] called attention to the fact that the ingestion of fluids is followed by an increase in the temperature of fevers. During fevers there is apparently a retention of fluid, and it is only upon defervescence that it is eliminated in quantity. The rise of body heat is in direct ratio to the accumulation of fluid in the organism; and Glax has found that a notable reduction of fever follows the withholding of liquids. His explanation is that the dilatation of the vessels which forms part of the febrile process permits the accumulation of water in the blood, thus favoring prolonged contact of the globules with the tissues and increasing oxidation. The facts upon which these conclusions are based require fuller investigation.

Semmola[21] prescribes glycerine diluted with water as a drink in acute fevers. The formula is as follows: Pure glycerine, 30 grams; citric or tartaric acid, 2 grams; water, 600 grams.—M. Of this solution twenty or thirty grams to be taken every hour. It proves a very agreeable beverage, well borne by the stomach and not followed by intestinal derangement. Its free use is followed by a notable reduction in the quantity of urea excreted.

Hare[22] has studied the influence of antifebrin, salicylic acid and carbolic acid on normal and abnormal bodily temperature. The objects of his research and the results will be found in the department of Experimental Therapeutics.

Finkler[23] observes that antipyretic treatment by baths must be given with reference to the laws governing nervous mechanism of heat and when thus given are of the greatest value. He believes that the drugs which directly influence fever have few grounds of real worth. He considers the combined use of baths and specifics as constituting the most efficient treatment.

Löwenberg[24] formulated the following general indications for the treatment of the infectious diseases:—

(1) To guard against further infection by the destruction of the infecting germs upon every accessible surface of the body and especially by the prompt evacuation and disinfection of the bowels; (2) an effort to eliminate, in part at least, pathogenic germs and their ptomaines by increased activity of the natural excretory

organs, as the intestines, the kidneys and the respiratory tract. (3) to increase the powers of resistance of the body and its organs by an abundant alimentation, by reducing the temperature by means of hydrotherapy and antipyretics, and finally by stimulation. In this connection it is probable that electricity will in the future prove of service.

Unverricht[25] formulated doubts which have been expressed by many clinicians, especially in Germany, as to the real benefit, not to say simply the curative influence, of antipyretic measures, and refers to Bauer, Pflügger, von Jaksch, Strümpell and Heubner as expressing doubts in regard to the positive views promulgated by Liebermeister concerning the disastrous influence of mere elevation of temperature.

W. P. Beall called attention to the fact that the dangers of high temperature in acute diseases may be overestimated. He believes that in the mere use of antipyretics we run a risk of paralyzing the heart.

Seay[27] regards the causes of fever as: (1) arterial contraction; (2) accelerated action of the heart; (3) increased tissue metamorphosis or oxidation; (4) probably a nervous influence causing an inhibitory action on the usual changes in transformation. He regards veratrum viride, aconite and gelsemium as valuable aids in the prevention of fevers.

J. R. Barnett,[28] of Neenah, Wisconsin, regards the salicylate of ammonium as among the more efficient of the antipyretics. As an antipyretic in fever marked by severe adynamia, it is among the safest on account of the ammonia base. It is stimulant as well as antipyretic, and therefore of itself fulfills indications otherwise only gotten by a combination of remedies. It is an agent of wide germicidal powers, being promptly efficient in affections of great etiological and pathological differences, each arising from its own proper specific micro-organism. As a remedial agent in typhoid and remittent fevers, it is unsurpassed, aborting them at the outset under favorable conditions, and greatly mitigating their severity and duration under circumstances less favorable.

Sullivan,[29] being favorably impressed with the theories and results presented by Barnett and Jackson, gave ammonium salicylate a careful trial, and draws the following concluions from his personal experience;—

"Ammonium salicylate is certainly a very effective antipyretic. It will not reduce temperature as rapidly as antipyrin or antifebrin, but the antipyretic effect is more lasting than that produced by either of these agents." He cannot agree with Dr. Barnett that it is a stimulant, but is inclined to believe that in large doses, or in moderate doses long continued, it has a decidedly depressing effect upon the heart and respiration. This depression may, however, be avoided by administering the ammonium salicylate in combination with the aromatic spirits of ammonia. It has an irritating action upon the kidneys, and consequently should not be given in scarlet fever, or in any case in which these organs are not in a healthy condition. He is accustomed to prescribe it in doses of eight or ten grains every two or four hours through the first day, then at longer intervals, as the requirements of the case indicate. In some instances, thirty grains given in divided doses during twenty-four hours caused decided ringing in the ears, while other individuals bore a drachm in the same time with but little disturbance. To children three years of age it may be given in three grain doses every four hours.

Sydney Phillips[30] insists upon the importance of keeping up so far as possible the force of the heart's action. He suggests that the albuminuria of fever may sometimes be the result of diminished arterial tension, since Charcot has shown that albuminuria may result from delayed blood-flow through the kidneys. Hyperpyrexia should be anticipated by antipyretic treatment, which is especially to be employed to secure intermissions or remissions in the course of the fever when they fail to occur spontaneously.

Da Costa[31] has directed the attention of the profession to the value of cocaine as a remedy in low fevers attended by nervous depression and weak circulation. Cocaine hydrochloride was given in doses of one quarter and even one half grain hypodermically every two hours. It is not clear from the account how long this treatment was kept up; but the results in his cases of enteric fever were very favorable. In this connection is to be taken the especial intolerance shown by many individuals for this drug, and the great nervous depression not rarely resulting from the use of small doses of it. Its administration must therefore in all cases be practiced at first with great circumspection.

Poulain[32] recommends for loss of hair after fever, frequent sponging of the scalp with one grain of tartar emetic dissolved in one fluid ounce of distilled water.

Valenzuela[32] used air containing an excess of nitrogen in the treatment of fever. He compared its action in reducing fever temperature with that of cold applications, quinine, antipyrin, digitalis and arsenic, and concluded that the inhalation of nitrogen is the only method of treatment which is not followed by a rise of the temperature to its former height upon its discontinuence for a day. He also regards it as having a favorable influence upon the morbid process itself. Nitrogen was administered in two daily séances of half an hour to an hour in duration, beginning with air containing 17.76 per cent. of oxygen and gradually decreasing this proportion until at the end of the sitting it was only 12 per cent. A single case of phthisis was reported in which the fever ceased after the use of this treatment. Other cases are alluded to as having shown its beneficial effects.

Valenzuela has also recently made a series of interesting observations on the power of one volume of pure oxygen to affect the febrile state. He found that the temperature of healthy rabbits, after being kept an hour in pure oxygen at pressure varying from 760 millimetres to 1520 mm'., underwent a marked fall, amounting in one case to as much as 11° F. Rabbits inoculated with septic material so as to induce pyrexia, suffered a marked fall of temperature by an immersion for one or two hours in an atmosphere of pure oxygen. In one experiment of two rabbits similarly innoculated, one which was bathed twice in oxygen of a tension seven times as high as that of the atmosphere, recovered, while the other, left untreated, died on the third day.

Mary Putnam Jacobi[34] concludes a very valuable communication on food and fever as follows:—

"The ideal quantity of nourishment in fever would be that which might entirely cover the waste of tissue caused by the breaking up of fixed albumen. But this ideal quantity can never be given, owing to the multiple disturbance of the digestive organs. The mucosa of the alimentary tract is hyperæmic, or inflamed, or ulcerated, and abnormal fermentations are constantly sustained on the surface. As a consequence, fever patients always suffer from digestive disturbance, of which the mildest is anorexia; but to this may be added nausea, vomiting and diarrhœa, of all degrees of severity. Food, therefore, in acute febrile diseases, must be prepared for speedy absorption: it must be liquid; and often it must be artificially digested. The meat solutions of Liebig and Rosenthal, and preparations of peptones, as that of Rudisch, are the most suitable forms of artificially digested albuminous food. Instead of sugar it is desirable to give glucose. There are no means of digesting fat, and this must be avoided in fevers, from its tendency to decompose into acrid compounds. The nutritive value of gelatine has been the subject of prolonged and learned controversy. It certainly cannot replace the fixed albumen of tissues, and thus is inferior to peptones, which have been shown to do so. But gelatine is a nitrogenous substance, which is destroyed in the organism with great rapidity, and gives rise to a proportionate amount of urea. It can therefore, to a certain extent, serve to spare albumen. One hundred grams of dry gelatine contain 17.3 grams of nitrogen; 163 grams of dry gelatine originates as much urea as 84 grams of dry albumen. In the formation of urea, physiological processes are instituted of at least temporary value in nutrition. These considerations justify the use of jellies and also of soups in fever. Inorganic salts are no unimportant part of necessary food. They are necessary to the assimilation of food by the tissues, and to the elimination of waste products from them. An excess is always eaten, and eliminated by the urine. The effect on osmosis is most marked with chloride of sodium, but is noticed with all salts. Bouillaud was in the habit of treating typhoid fever patients—having first bled them largely—almost exclusively with table salt. In large doses this substance has some degree of antiseptic action in the intestines. Mucilaginous drinks are always suitable in fevers, although it be unnecessary to ascribe to them the traditional virtues of the French tisane. Alcohol is to be considered rather as a food than a drink. In small doses alcohol is certainly decomposed and oxidized like any carbohydrate substance; thus, in doses of four ounces and less in the twenty-four hours. Above this limit, with considerable margin of variation in the individual case, alcohol is eliminated unchanged; and this quantum, as a special stimulant of the heart and nerve tissue, is therefore a medicine and not a food."

Francis Duffy,[35] in an essay on alimentation in continued fevers, to which the Medical Society of the State of North Carolina awarded a prize, makes an admirable plea for moderation in fever feeding. It is only in health that the fundamental processes of nutrition—viz., digestion, absorption and assimilation—are completely performed. The idea that the increased destructive metamorphosis incident to the fever process calls for increased nutrition would, from a superficial view, appear to be theoretically correct; but as a matter of fact it is impracticable. The rapid molecular death of the tissues under the influence of high temperature and the complexus of chemical and vital phenomena which make up the fever process, is incompatible with great tissue-building to an almost prohibitory degree and the digestive laboratory is crippled and incapacitated for performing its functions. We cannot rebuild the new edifice while the old one is yet in flames. If, when gastric digestion is suspended, albuminoid substances are artificially converted into peptones, absorption is not assured. If accomplished on the principle that any liquid would be taken up by a thirsty soil, the other, and crowning act of nutrition—that is, assimilation—can only be performed in a limited degree; and the blood, already burdened with waste products of the body, is further charged with the disposal of the excess of alimentary principles. Retrograde metamorphosis goes on in the blood, and the amount of urea excreted represents not only the tissue waste, but also the waste of alimentary principles,—peptones not converted into tissues. He regards the large amount of milk administered to fever cases by many clinicians, and particularly by Loomis, as injurious, and agrees with Fothergill as to the advisability of using the carbo-hydrates rather than the albuminoids. In deference to Fothergill's advocacy of grape sugar and glucose, as well as to the desires of fever patients, he has used the juice of ripe grapes, and has had on no occasion cause to regret it. Food should be administered only in such quantities as are capable of digestion and assimilation. If a patient without the power of digestion has a conservative loss of appetite, is it not equally true that a patient without powers of assimilation has a conservative loss of digestion?

Dujardin-Beaumetz[36] follows a discussion of the historical aspect of the question of alimentation in fever by practical observations on the teaching of modern science. One of the first consequences of fever is a diminution or even a chemical change in the digestive secretions. In fever the digestive apparatus is diseased and the intestinal juices are much diminished. The peptonization of albumen and the emulsification of fats is not properly brought about. Liquids are capable only of resorption. Fever patients invariably lose weight. The loss takes place through the excretory organs, the lungs, intestines, kidneys and skin. As the resorption of any thing but liquids and fats is almost nil, the first rule must be to give fever patients nothing but liquids. Milk and bouillon are most available. The water and salts undergo a ready absorption. The fate of the albuminoids and fat contained in the milk has not yet been determined; it is probable that they are little if at all used. As regards alcohol, in the case of children, old people, and drinkers, its administration in the form of wine is often useful and sometimes indispensable. As regards its physiological value, there is much difference of opinion. According to some it increases the vital powers; according to others it reduces temperature; according to others again, it acts as a sparing agent; and lastly some authorities regard it as a food. It is possible that it acts to some extent in all these directions. These, then, are the proper articles for the alimentation of fever patients,—milk, bouillon and wine. Dujardin-Beaumets, does not, like Nothnagel, recommend broths, soups and eggs, nor is he in favor of such an early return to solid food as are the Germans. Patients, he thinks, should not be allowed the use of solid food in the period of convalesence so soon as they become eager for it.

Nevin[37] regards the proper treatment of fever as purely dietetic, and in view of the part played by the nervous system in the mechanism of fever, as purely calmative. Under the latter heading are to be mentioned good nursing, the removal of irritation and means to lower excessively high temperature.

ENTERIC OR TYPHOID FEVER.

Etiology.—Brouardel[38] has communicated to the Academy of Sciences the details of a localized outbreak of typhoid fever in which it is claimed that the specific bacillus was found in the contaminated water. The facts are as follow:—

During the months of August and September, twenty-three persons had occupied three contiguous houses at Pierrefonds, of whom twenty were attacked with typhoid fever. Four members of one family died. Insanitary conditions of the most aggravated kind existed. The water supply was from shallow wells filled by percolation from a neighboring stream. One cess-pool was situated at a distance of thirty feet from a well, the other at a distance of sixty-five feet. The soil was light and porous, and the level of the water was below that of the cess-pools. The interesting feature of the investigation was the detection, by Chantemesse, Director of the Bacteriological Laboratory of the College of France, of the bacilli regarded as the specific agent of typhoid fever. On October 18th they were present in large numbers in the water supplied to the house in which the four deaths had occurred. On October 29th there were fewer, and on November 21st none could be found. In the other wells similar organisms did not exist.

To demonstrate the identity of the bacilli found in the water with those of the typhoid patients, the spleen of one was punctured with a sterilized trocar on the tenth day of the illness, and in cultures of the material colonies developed the same morphological and biological characters as those obtained from the well water. The coloration and sporulation of the bacilli were identical.

Vidal and Chantemesse[39] have succeeded in carrying the researches on the bacillus of typhoid fever rather further than Gaffky, who described it. The central clear space, they say, is not characteristic, as Artoud supposed; for it is found in other bacilli, especially in those of old cultures, and it is, they believe, the beginning of the death of the microbe. Spores are produced between 37° and 38° C. It does not liquefy gelatine, but is easily cultivated on potato. Gaffky was unable to find the bacillus in the living subject or to inoculate it. Vidal and Chantemesse have found it during life by making capillary punctures of the spleen, and they have been able to inoculate both mice and guinea pigs, and have subsequently found the bacillus in the abdominal viscera and lungs. In a case where a typhoid patient aborted at the fourth month, the bacillus was found in the placenta. Hallopeau,[40] Lardier and others contributed reviews of existing knowledge upon the same subject.

Fränkel[41] reported a case in which a patient who had had typhoid fever, followed by relapses at intervals of six weeks and four months, developed some time afterwards an abdominal tumor. Upon aspiration a quantity of pus was evacuated, and in it were found typhoid bacilli, the cultures of which were characteristic. He regarded the case as one of inflammation produced by the persistent typhoid bacilli, which had failed to undergo attenuation at the close of the initial paroxysm, as is usual.

Wiltschur[42] has tested the action of boiling water upon cultures of typhoid bacilli. He finds that twice the volume of boiling water will destroy the bacilli, and three times their volume will destroy their spores; that from two and a half to three times their volume destroy the bacilli in stools, and that four times their volume is sufficient to destroy their spores.

A recent epidemic of typhoid fever at Mount Holly, N.J., has been ascribed to improperly constructed cess-pools at a settlement called Smithfield, which contaminated the Rancocas River, from which the water supply of Mount Holly is derived.[53]

Pathology.—"In an Amsterdam graduation thesis, by Dr. M. Neimeijer, on the Statistics of Typhoid Fever, out of 50 cases in which complete observations were recorded, prodromata occurred in 25, rigors in 16, "cold shivering" in 1, pain in the left side in 7, diarrhœa in 42, splenic enlargement in 48, rash in 47, ileo-cœcal gurgling in 19, and pain in the same region in 20, bronchial catarrh in 43, albuminaria in 12, in 3 of which cystitis followed. In 63 cases, relapses occurred 6 times. With regard to complications, out of 73 cases, intestinal hæmorrhage occurred in 5; peritonitis in 3, perforation occurring in 2 of these. Other complications were: Pharyngitis, 1; acute follicular sore-throat, 1; parotitis, 1; epistaxis, 3; laryngeal perichondritis, 1; pulmonary infiltration, 13; pleurisy with effusion, 3; thrombus of the crural vein, 1; acute nephritis, 1, in a somewhat doubtful case; nephrolithiasis, 2; herpes labialis, 3; cutaneous hæmorrhage, 3; periostitis of the tibia, 1; joint-affections, 3; meningitis, 1; neuralgia of the sole, 1; profuse perspiration, 2; polyuria, 1; mental disturbance, 4; deafness, 5; bleeding from the ear, 1. The total number of patients on whom observations were made was 194. Of these, 20, or 10.3 per cent. died, the male mortality being decidedly higher than that occurring among female patients,—12.5 per cent. as compared with 6 per cent."

Willememminger[45] reports a case of typhoid fever in a girl six years old in whom about the beginning of the second week, characteristic symptoms of measles showed themselves. The case ran a favorable course.

This case is open to criticism. The editor of this department has on several occasions seen the characteristic rose-rash of typhoid fever so abundantly developed in childhood as to lead an incautious observer to make the diagnosis of measles.

Jencks[46] reports a case of typical typhoid fever, and in a general discussion of the subject quotes a lecture by J. C. Wilson in which the atypical forms are classified as follow: (*a*) mild typhoid; (*b*) abortive typhoid; (*c*) typhoid of childhood [infantile remittent]; (*d*) typhoid of the aged; (*e*) cases of febrile intestinal catarrh, (*f*) cases of afebrile intestinal catarrh.

Mettler[47] considers gangrene occurring as a sequel of typhoid fever to be due to mechanical obstruction of the arterial branches, producing dry gangrene, or of obstruction of the artery and vein producing moist gangrene by coagulation of the blood. This coagulation, while favored by causes of a mechanical nature, is essentially due to endarteritis. The local treatment of endarteritis of typhoid fever consists in elevation of the limb, the application of leeches followed by hot stupes or if more agreeable to the patient, cold water dressings, poultices of chamomile flowers or decoction of poppy heads. The circulation should be supported by means of alcohol and digitalis. The peripheral circulation should be stimulated by alternate applications of heat and cold. For this purpose the constant current battery may be advantageously used. After all efforts, it may become necessary to make numerous incisions into the mortified tissues and follow by proper surgical dressings.

R. S. Archer[48] reported an interesting case of brachial monoplegia complicating enteric fever. Tissier[49] contributed an important paper upon the laryngeal complications in typhoid fever to which is added a full bibliographical index.

Weiss[50] reported the case of a soldier aged 22 years who died from profuse gastric hæmorrhage about the beginning of the third week. This had been preceded by intestinal hæmorrhage. The diagnosis of hæmatemesis due to the anatomical lesions of typhoid fever was based upon the following facts: At the time of the attack of fever, the patient was in excellent health and had never suffered from gastric disease. He was not a drinker, had never had syphilis, and the heart and lungs were sound. There were no petechiæ in the skin, and there had been no epistaxis; and, finally, a careful investigation of the naso-pharyngeal cavity revealed no lesion whatever. The source of the fatal hæmorrhage was therefore the duodenum, the lower portion of the œsophagus or the stomach, and its cause, considering the previous physical condition of the stomach, was presumably typhoid ulceration.

Many observers have testified to the occurrence of typhoid ulceration in the stomach and its immediate neighborhood. Louis and Jenner observed typhoid ulcerations in the lower portion of the œsophagus. Röderer and Wagner described an œsophagitis follicularis, and Eichhorst and Reimer observed similar changes. Rokitnasky noticed infiltration and even necrosis of the lenticular glands of the stomach, and Chauffar and Cornil described an infiltration of the mucosa and sub-mucosa with lymphoid cells, which may proceed to the formation of miliary abscesses.

Diagnosis.—Masse[51] reviewed *in extenso* the present state of knowledge of typhoid fever as observed in the French possessions in Africa. The following table of the differential diagnosis between typhoid fever and the remittent or pseudo-continuous fever of the tropics is of importance:—

TYPHOID FEVER.	REMITTENT FEVER.
Commemorative.	
No previous exposure to marsh miasm. The patient is usually between 18 and 25 years of age. If in the service, he is a new-comer. If in civil life, he has recently changed his abode. Season of the year, indifferent.	Previous exposure to marsh miasm. Age indifferent. Attack either vernal or autumnal.
PRODROMES.	
Prodromes usually present. Loss of strength, indisposition to ordinary occupation, sense of fullness in the head, epistaxis, diarrhœa, loss of flesh, restless nights, insomnia alternating with disagreeable dreams, gastric derangement followed by headache, malaise, transient fever, vertigo and tinnitus aurium.	Prodromes sometimes present, sometimes absent. When present, of short duration. Gastro-hepatic disturbances, headache, exhaustion accompanied by nausea, sometimes by vomiting. When there is prodromic fever, it appears during the day and is quotidian or double quotidian in most cases. Occasionally there is diarrhœa.
ONSET.	
For several days, slight febrile movement with evening exacerbation. An	Onset sudden, without previous fever except perhaps transient paroxysms

abrupt onset with shivering followed by pyrexia, is rare.

occurring during the day with complete intermission. The onset is, as a rule abrupt, accompanied with shivering, headache and active febrile movement.

THE DEVELOPED ATTACK.

Facies pale, dull, depressed, rarely red and animated. Headache active, persistent, often retro-orbital. Dizziness and tinnitus, epistaxis, mental hebetude, subdelirium or delirium during the night. Drawling speech. Tremulous tongue and lips. Pulse frequent. Temperature 38° C. to 40° or 41° C. Gradual rise, reaching the fastigium by a series of oscillations which occupy from three to six days, the temperature of the evening exceeding that of the following morning. Tongue coated in the centre, red at the point and borders. Abdomen full and tympanitic, chiefly so in the hypogastrium. Tenderness upon pressure in the right iliac fossa, where there is also frequently spontaneous pain. Borborygmi. Rose spots. Hypertrophy of the spleen ordinarily difficult to recognize in the first days. Diarrhœa as a rule, constipation is the exception. Thirst, anorexia. Nausea and vomiting unusual.

Sibilant and sonorous rales on both sides of the chest, more distinct posteriorly.

Gradual progress of the sickness. Sudamina appear toward the end of the second week. Ataxic accidents, as delirium, coma, subsultus, adynamia, etc., together with stupor also occur toward the end of the second week, sometimes later. Grave disturbances of nutrition, rapid wasting.

Ten or fifteen grams of the morning urine treated with four or five grams of nitric acid, shows at the bottom of the glass an indigo diaphragm, often accompanied in grave cases by a second diaphragm, which is white composed of albumen and separated from the first by a bed of urine more or less thick. Sometimes there is a third diaphragm, which is also white and formed of uric acid and separated from the diaphragm of albumen by a bed of urine.

Face injected or of a somewhat earthy hue, rarely pale. Intense headache, likewise often retro-orbital. Vertigo, tinnitus and epistaxis, all rare. Intelligence more acute. Delirium and subdelirium rare. Speech more natural. Absence of the trembling of the tongue and lips. Eyes and external integument icterode in bilious remittent. Pulse frequent, not dicrotic. Temperature 38° to 40° or 41° C. Course of the fever irregular and interrupted, with abrupt rises and incomplete remissions, the temperature of the morning often being higher than that of the evening. Tongue moist and white in the centre and red at the borders; covered with a yellowish coating in bilious remittent. Abdomen prominent, principally in the hypogastric region. Tender upon pressure in the epigastric and hypochondriac regions, and above all in the left hypochondriac. Borborygmi, rare. Rose spots, absent. Hypertrophy of the spleen difficult to recognize in the early days. Diarrhœa rare in the ordinary form of remittent, very common in bilious remittent. Constipation the rule. Great thirst, nausea, and in bilious remittent, vomiting.

Bronchial râles rare.

Course rapid. No sudamina, as a rule, but when present they appear in the first week. Stupor and ataxic accidents when they occur are early. Nutrition less profoundly disturbed. Emaciation moderate in ordinary remittent, but marked in bilious remittent.

The urine treated in the same manner, shows at the bottom of the glass a brown diaphragm. In the more serious cases, the brown diaphragm is found accompanied by a diaphragm of albumen from which it is separated by a bed of urine more or less thick. In a pronounced case of bilious remittent, there is formed with or without the diaphragm of albumen, a diaphragm of the coloring matter of the bile. Sometimes there is here likewise a diaphragm of uric acid.

TREATMENT.

Inefficacy or slight utility of the sulphate of quinine in uncomplicated typhoid fever. Convalescence slow.	Very great utility of the sulphate of quinine. Convalescence rapid in ordinary remittent. Less rapid in bilious remittent.

PATHOLOGICAL ANATOMY.

Tumefaction and ulceration of the Peyer's patches and solitary follicles. Enlargement of the mesenteric glands, etc., etc.	Not the least tumefaction or ulceration of Peyer's patches or Bruner's follicles. Mesenteric glands unaffected.

Masse concludes his paper with the following considerations regarding typhoid fever in Africa, according to historical periods:—

First Period.—In the earliest days, inflammatory fevers, typhoid fever and remittent fevers were frequently confounded, and were submitted to antiphlogistic treatment. This period is characterized by an abuse of general and local blood-letting in the treatment of these pyrexias.

Second Period.—This period began in 1836 and continued several years. It is the natural outcome of the reaction against the antiphlogistic treatment, in consequence of a closer study of fevers of paludian origin, and of a nicer appreciation of the nature of fever itself and the treatment applicable to it. The name of Maillot is inseparably connected with this period. The rational views of this period were distorted by the exaggeration of the influence attributed to paludism. There were only pseudo-continuous fevers, and typhoid fever disappeared, so to say, from the nosology of Africa. The abuse of the sulphate of quinia is characteristic of this period.

Third Period.—This we gladly name the scientific period. Typhoid fever is again recognized. It is studied methodically and compared with the dothienenteric of France, and found to be the same thing. An analysis of their respective symptoms shows remittent and typhoid fevers to be essentially different maladies. Sulphate of quinia largely used in remittent is less employed in typhoid, and then only to meet precise indications.

Present Period.—Without wishing to detract from the great utility of the discovery of the microbes of malaria from the standpoint of the differential diagnosis between the two forms of fever, we venture to say that in practice the diagnosis will be made in accordance with the precepts of traditional medicine. In fact the serious and attentive study of the symptoms of each sickness, their order of appearance, and their relative value, the course and the nature of the affection itself, the etiology, etc., must always form the positive basis of an exact diagnosis from which alone can be derived the elements of a differential diagnosis.

According to G. Harrison Younge,[52] in India the rule is always adopted of suspecting every case of continued fever to be one of enteric fever until it can be proved to be not so.

Spillman[53] contributes three cases of enteric fever in the course of which herpes labialis was observed. These cases disprove the view at one time held that the eruption in question did not occur in the course of enteric fever, and that its appearance in any obscure case precluded the diagnosis.

J. C. Wilson[54] reported a case of enteric fever in a precociously developed girl at the age of puberty, in which death occurred at the end of the first week from intercurrent fulminant peritonitis of pelvic origin.

At the autopsy the lesions of both of the above conditions were found. The peritonitis resulted from acute double septic salpingitis.

Money[55] confirms the assertion of Hughlings-Jackson that the knee-jerk, as far as his experience goes, is never absent in typhoid fever, whilst in tubercular meningitis its disappearance for a few hours or a day or a few days is by no means rare. A variable state of the knee-jerk, that is one day present, the next absent and the third increased, points to meningitis and not to typhoid fever. He makes what may be considered a curious comparison, and asserts that the differential diagnosis between meningitis and the essential fevers may be assisted far more by a study of the knee-jerk than by swelling of the spleen. For unless the splenic enlargement be enormous, tubercle as well as typhoid fever and other diseases—as, for example, pyæmia—will account for it; whereas absence of knee-jerk, or rather ascertained disappearance of the same, is probably never met with as the simple outcome of prolonged pyrexia.

I. E. Atkinson[56] presented a valuable paper on forms of typhoid fever simulating remittent malarial fever, and demonstrating the essential typhoid character of many common forms of fever not usually recognized as such. He considered it safe to predict that the solution of diagnostic difficulties will be readily obtained through the rapidly increasing knowledge derived from bacteriological researches and culture observations. In the discuscussion of this paper, Councilman and Osler agreed with the author that we have in the microscopic examination of the blood a positive means of diagnosis.

Prognosis.—Bardat de Lacaze[57] discusses the subject of sudamina in typhoid fever especially from the point of view of prognosis. Sudamina are common enough in typhoid fever, as in other maladies attended by copious perspiration. Murchison, who observed sudamina in about one-third of his cases, attributed no special significance to them. In this he has been followed by most authorities. De Lacaze holds that the appearance of sudamina or malaria in the third week in severe cases is of favorable omen; that in a day or two the temperature will fall. Like eruptions appearing earlier, that is, during the second week, are of less significance.

Treatment.—Von Ziemssen's lectures on the treatment of typhoid fever, contain the following recommendations:—[58]

The room should be quiet, large and easily ventilated, and have a small room adjoining in which baths can be given. It is important that there should be a second bed in the room standing near the patient's bed, into which he can be lifted when his bed is soiled or must be rearranged. Frequent change of bed is especially gratifying to most patients. The nurse should be trained, accurate, reliable and uniformly quiet. The temperature should be taken by a skillful hand. Nursing of the patients by female relatives is decidedly objectionable. No visitors should be admitted until convalescence is far advanced. The patient should not be allowed to move himself or get out of bed. Sudden death frequently results from a neglect of this rule. The greatest order and cleanliness should prevail in the sick room. A water mattress is indispensable in severe cases, especially if bed-sores threaten. The back should be rubbed once or twice daily with spirituous solutions, weak warm rum, cologne water, etc.

Dietetics constitute the most important part of the treatment. No pure albuminous food and still less fatty food should be given. We must adhere as closely as possible to the normal proportion of food stuffs for the healthy organism as given by Voit, with the special consideration that fats are taken with difficulty by fever patients, best in milk, and that carbo-hydrates are not only best taken but best borne and markedly limit the consumption of the albuminous and fatty matters of the body. Frequent change and variety of flavor and consistence of food is desirable. The diet should be liquid. Starch, dextrine, and sugar may be given in various forms, both in food and drink. Thin oat and barley water are recommended. The flavor is varied by the occasional addition of sugar, cinnamon, wine, etc. Clear meat broths with or without the yelk of egg, meat extracts, etc., are available. Milk is the most complete mixture of albumen, fat and carbo-hydrates and is an excellent food in typhoid fever, but too much of it will cause uneasiness in the stomach. Von Ziemssen does not give more than one pint as a rule in the twenty-four hours. Freshly expressed meat juice, which consists, in fact, of serum, lymph and blood, forms an acceptable and highly digestible food. Of this about 150 or 200 grams may be used in the 24 hours. It may be given partly in spoonful doses like a medicine or partly admixed with soup, the temperature of which is not above 46° C., that is to say, 114.8° F. Frozen meat juice is also well borne. If eggs are administered in the soup, not more than three should be administered during twenty-four hours. A very judicious combination is yelk of egg with brandy. Light non-acid white or red wines are used from the beginning of the sickness to the extent of about one pint daily, Mulled wine may be given, tea with rum or brandy toddy, also jellies prepared from fresh calves' feet, with white wine. On account of their refreshing and pure taste they are very grateful to the patient. As a rule the food and stimulants must be proportioned to the severity of the disease. Food may be given every two hours, and drink and medicine between times. Feeding must be kept up through the night as well as the day. The diet of convalescence should follow the usual diet of health with certain modifications. The return to solid food should be as follows: First breakfast, tea with biscuit and one soft boiled egg. Second breakfast, 100 grams of finely minced raw bacon with wheat bread crumbs. Noon, 150 grams pigeon, young chicken, or partridge finely minced in soup. Later with a mild sauce and mashed potato and with wine or beer. In the afternoon, tea with biscuit or cakes. For supper mush and milk, two soft eggs and some raw bacon. This may soon be followed by calves' feet for breakfast, then an English broiled beefsteak, mutton and preserves, and in the evening some strengthening soup and some beer. At this stage, when the patient thinks of nothing but eating, something new in the way of food must be given daily.

The only drug to which we can ascribe a kind of specific action is calomel, perhaps on account of its entire or partial change by the chlorine of the sodium chloride in the intestinal juices, into corrosive sublimate. Von Ziemssen gives calomel in three doses of gram. 0.5 in two hours and then obtains a number of thin grass- or moss-green calomel stools and considerable reduction of temperature, which often goes down to normal and remains there about twelve hours. These are the primary effects of the calomel. The secondary effects of the drug consist in modifying the intensity of the infectious condition, as has been shown by Liebermeister. The earlier calomel is given, the better,—best at all events within the first five days, and it should be used during the first week. Its action is very possibly a sterilizing one on the specific bacteria vegetating in the intestine. The abortive action of calomel, such as has been claimed by Wunderlich, has not been observed. Griesinger, Baumler and Weil have shown that neither the mortality is lessened nor the number of abortive cases increased by its use, and that no other properties belong to calomel than those of a mild non-irritating evacuant which reduces temperature. There is no question of mere eccoprotic action, since the effects upon the temperature and the local intestinal affection are observed in cases where active diarrhœa already exists. Attempts to fix the hypothetical specific action by long continued calomel treatment and to force a true abortive calomel treatment have at different times failed, as has also the sublimate treatment of typhoid fever.

Next to calomel, baths deserve the greatest consideration. Von Ziemssen first gives a warm bath to cleanse the skin and remove parasites. The temperature is then observed by bi-hourly rectal measurements for ten to twenty-four hours to determine its course. Lukewarm baths of 86° F. to 81.5° F. of fifteen minutes' duration, with continual stirring of the water and washing over the upper part of the body so far as it is not dipped in the water, are then commenced. The higher the temperature, the more severe the cerebral affection and muscular weakness and the intestinal affection, so much cooler must the bath be. For several years Von Ziemssen has not given a bath below 63.5° F. The bath-tub is placed by the patient's bed with a screen around it and the patient is lifted into it. The water is renewed once only in twenty-four hours. In many cases, the bath is given only lukewarm, not under 75° F. during the whole course of the sickness, beginning at a temperature of 88.2° F. to 86° F. While the patient sits in the bath, the temperature is reduced to 75° F. or 72° F. by the addition of water to the foot and continual stirring. Such gradually cooled baths are especially recommended for patients of a weak, hyperæsthetic and nervous constitution, and especially for women and children. For young, robust and vigorous constitutions the simplest way is to use at once the cold bath of 63.5° F. to 65.5° F., as Vogl and his military colleagues do with their soldiers. It is in the highest degree important to vary the temperature of the bath according to the constitution of the patient, the stage of the disease, the time of day, the patient's temperature and the condition of the nervous system. In general terms, the following rules are to be observed: the earlier the stage, the higher the fever. the more robust the constitution, so much the colder should the water be; on the other hand the later the stage, the weaker the constitution, the more affected the nervous system, the warmer should be the water. Routine directions are bad in practice.

In regard to the frequency of the baths during the day and the time at which they are to be given, authorities are not in accord. Some bathe patients from ten to twelve times in 24 hours, others from two to four times only. Many prefer to give baths at night, others by day and night, and the majority of physicians only during the day. Von Ziemssen is in accord with Liebermiester in holding that baths have a better general effect when given during the normal temperature remission than during the period of exacerbation. Von Ziemssen thinks that three or four baths in the 24 hours are usually sufficient, though there are cases in which six to eight baths must be given. There are milder cases in which only one or two baths are necessary. Children should have less frequent and warmer baths than adults.

The duration of the bath should, as a rule, be not less than fifteen minutes, nor more than thirty minutes. The signal for its termination should not be a mere sensation of chilliness, but actual shivering. Von Ziemssen thinks that other hydrotherapeutic measures which have been recommended as substitutes for douches and complete baths—as moist cold, the cold pack, sponging with cold water or with a mixture of water and vinegar—have generally no other value than that of a temporary refreshing of the patient, and are almost wholly without influence upon the temperature and the objective state of the nervous system. Spontaneous morning remissions are the signal for lessening the number of baths and increasing their temperature.

Von Ziemssen formerly used quinine in large doses as an internal antipyretic, but he has recently abandoned it wholly and uses antipyrine instead as being, in divided doses amounting in all to gram 5, much more certain and entirely free from the undesirable after-effects of quinine. The temperature falls from 2 to 4° C. and remains low for some time, certainly as long as after the administration of massive doses of quinine. The rubeoloid eruption frequently observed after its administration is of no significance. It may also be administered in clysters. Thallin, while exerting a distinct antipyretic effect, can not take the place of antipyrine. Antifebrin is also a valuable antipyretic, having in its favor the smallness of the dose, which is about one-fifth or one-fourth that of antipyrine, and its cheapness.

Management of individual symptoms and symptom excesses plays an important part in the treatment of typhoid fever. Cerebral symptoms, headache, oppression, vertigo, call for the prolonged use of ice bladders. In those patients to whom permanent cooling of the head is unpleasant, the subjective sensations must be taken into consideration. Sleeplessness, restlessness and tossing at night are best controlled by moderate doses of morphine. Very active delirium yields to the use of lukewarm or warm baths in the evening or early in the night. If these are not adequate, hypodermic injections of morphine should be employed. In graver cerebral troubles, cold baths and very cold douches are better.

Cardiac weakness is of most dangerous significance and demands prompt attention. In the treatment of this condition, camphor is preferred; one part of camphor dissolved in five parts of olive oil may be administered hypodermically, two to five syringesful at a time. Of wines, Von Ziemssen prefers champagnes, and of concentrated alcoholic stimulants, he gives preference to cognac. The quantity to be given in twenty-four hours must be graduated according to the indications and the effects of the first doses. Too much is better than too little.

Bed-sores may be absolutely prevented by a large water mattress. When they occur they should be dressed with boracic acid salve, zinc or lead plaster mulls and white lead plaster. The patient, even if lying on a water bed, should be turned on his sides for a few hours each day. Furuncles, circumscribed gangrene, etc., rarely or never occur under antipyretic treatment. Severe intestinal affection and excessive meteorism may be ascribed in the majority of cases to faulty diet or the frequent use of opium and tannin. Excessive meteorism is attended with great danger. For its relief, ice bladders to the abdomen or Priessnitz's pack are advised. Small doses of calomel as recommended by Friedreich, or naphthalin or turpentine in clysters. If the diarrhœa does not exceed four or five stools in the 24 hours, it demands no treatment. If it exceeds this, it may be treated by small clysters containing tincture of opium. If intestinal hæmorrhage occur, every kind of food, all medicine, baths and every movement of the body are discontinued, ice bladders placed over the abdomen, and thirst quenched by small bits of ice. A syringeful of solution of sclerotic acid in water, one part to five, may be given every half hour as a styptic. In very profuse hæmorrhage, threatening life, enemata of ice water are used. When the hæmorrhage is very profuse and death from cardiac paralysis is threatened, injections of blood or salt water may be given. Bleeding during the period of late healing, fourth to sixth week, is more grave as regards prognosis than during the period of sloughing, in the second or third week, because in these cases, it is almost always due to delayed healing of the ulcers. Opium by the mouth and by the rectum in large doses is advised in cases of perforation, or injections of morphine may be used. Ice bladders should be placed over the abdomen and no food at all should be given. The thirst may be quenched by small pieces of ice, and every movement of the body avoided. Should sacculation of the perforation occur the sacculation should be opened and treated antiseptically. Puncture of the abdomen with a thin trocar to reduce the tension in the peritoneal cavity is only of temporary benefit. Von Ziemssen does not refer to the surgical treatment of perforative peritonitis. The treatment of complications such as pneumonia, erysipelas, parotitis and otitis must be regulated by the accepted rules for the treatment of those processes. Prophylactic treatment consists in the use of antiseptic washes for the nose and mouth.

The care of convalescents from typhoid fever must be carried out with great strictness. The treatment of relapses is the same as that of the primary attack, but less energetic. A month or two after convalescence has been fully established the patient should be sent away to the mountains or a climatic resort, and where circumstances permit he should have complete rest for a year.

Nothnagel[59] considers the indications for the treatment of typhoid fever to be: 1. Indicatio causalis. 2. Indicatio morbi. 3. Indicatio symptomatica. As regards the first, nothing is to be said. The indicatio morbi would consist in having a remedy capable of destroying the bacillus of typhoid without destroying the individual. Nothnagel takes a decided stand as regards the treatment of the acute infectious diseases upon the point that we must search for causal remedies. In quinine we have had a specific remedy for malaria for the past two hundred years. We know that quinine renders the malarial poison innocuous without damaging the organism. If syphilis depends, as it seems to do, on an infection by bacilli, we know that mercury and iodine gradually destroy the syphilis bacillus without the organism being destroyed or damaged. The task of therapeutics as regards the acute infective diseases is to search for specific remedies; but as yet they have not been found. Calomel has been recommended by numberless authorities. If it is given as an abortifacient in typhoid, it must be given in the first week, when the diagnosis is as yet only probable. A milder course, not a cure, and a shortening of the disease are obtained, according to Weil. Nothnagel has not been able to convince himself that this is the fact. After comparative investigations, he has gained the impression that castor-oil acts exactly like calomel. Those observers who contend for the abortive action of calomel, hold that it becomes converted in the intestine into sublimate, and in this way acts upon the bacillus. But this has not been proved, and we cannot yet regard calomel as a specific.

When we come to consider the symptomatic treatment, the three indications that meet us, even in uncomplicated cases, are (1) the administration of a sufficient amount of nourishment; (2) the reduction of temperature; and (3) the keeping within bounds of abnormal secretions. We must give our patients sufficient food, and food in a fluid form to secure its digestion. Water takes the first rank among the foods. It is so much more necessary to give patients water when they are insensible, and do not themselves request it. It must be given regularly; every quarter of an hour some milk or water, or wine or the like must be given. The water must be good and pure, and is best boiled. Nothnagel warns against the use of aerated waters. Bouillon is not a food. If it is given at all it should be used only as a vehicle for real foods. Patients get daily from a litre to a litre and a half of pure boiled milk, which may be taken either warm or cold. If they object to pure milk, a little tea, or a few spoonsful of brandy are added. Besides the milk, the yelks of from four to six eggs are given, and if soup is given the whites of eggs may be stirred up in it. Then the yelk of egg may be mixed with bouillon, chicken broth, pigeon broth, beef tea, veal broth, or gruels of oatmeal, rice, barley, etc. Such foods must be given at intervals of one half to two hours. The food is not changed until two or three days after defervescence is completed; then calves' brains, or sweet breads may be given, cocoa, or chocolate with egg, and biscuit. Six days after the subsidence of fever, meat may be given,—if the exhaustion is very great, a little earlier than this. It must be carefully prepared, raw or scraped, with salt added to it. A coffee-spoonful at a time. The slight rise in temperature that follows the first administration of meat is usually not important.

A very important point in the treatment consists in the administration of alcohol, which acts in the acute febrile diseases as a preventer of waste, the alcohol itself undergoing oxidation more readily than the bodily material, therefore being burnt up in the organism whilst the bodily tissues are spared. In this way it acts directly as a food for the preservation of the organism. Nevertheless, Nothnagel is decidedly opposed to the promiscuous exhibition of alcohol without discrimination in acute febrile diseases. It is further useful as a cardiac stimulant. In typhoid fever where we have a long-continued febrile process, it is requisite and necessary to give wine in all cases, without exception, from the first. The quantities that may be administered are enormous, delicate ladies not accustomed to wine having taken in a day a whole bottle of wine containing three-fourths of a litre, of which 10 per cent. is alcohol, ten to sixteen coffeespoonfuls of brandy and some beer.

In regard to the second indication, the combating of the fever. the views that are held by the majority of German physicians on the treatment of typhoid fever may be summed up as follows:—

A decided antipyresis is necessary in hyperpyrexia. It is unconditionally demanded in typhoid when morning and evening temperatures keep above 40° C., that is to say 104° F., and when decided nervous systems are present. So long as the patients have a clear mind and a morning temperature of 39° C. with a distinct pulse, antipyresis is not unconditionally demanded.

Antipyretics may be divided into two great groups: the chemical antipyretics.—those which reduce temperature by a chemical action; and hydrotherapeutic measures,—those which reduce temperature by the abstraction of heat. Formerly quinine was the only known chemical antipyretic. In the last few years, after a number of these drugs have been tried and abandoned, we have become acquainted with thallin, antipyrine and antifebrin. All these remedies are capable of reducing temperature in a remarkable manner. When a crisis arrives in an acute febrile affection, it runs its course in the great majority of cases without injurious complications: the temperature falls, the pulse slows and the patient feels well as the disease comes to an end. But there are cases in which the crisis takes place with injurious and dangerous accompaniments: symptoms of collapse comes on, the temperature and pulse fall to below normal, and the pulse rate becomes arhythmic. Cerebral symptoms make their appearance and are dependent upon anæmia of the brain: these are designated the delirium of inanition, and the coma of inanition. Such antipyretics as resorcin and kairin reduce the temperature energetically, but are followed by unfavorable symptoms. But the three new antipyretics above mentioned only rarely give rise to unfavorable symptoms. Nothnagel thinks that the three are of equal value, but has a certain paternal predilection for thallin. No one method of antipyresis is the most applicable. We must individualize our cases. If we would abstract heat in a case of typhoid, various methods are at command,—baths, packs and sponging. Packs are employed mostly in the case of children. The baths, however, constitute the principal method, and are sometimes administered warm and sometimes cold. The cold baths are given at a temperature of 22.5° C.,—that is 73° F. The patient is left in such a bath from three to seven minutes. In the case of weak persons, a little wine must be administered before and just after the baths, and hot water must afterwards be put to their feet. When the patient begins to shiver, he must be taken out. When he can not bear such a low temperature he must be put in a warmer bath, the temperature of which is gradually lowered. The warm baths are administered at a temperature of 86° to 90° F., and the patient is allowed to stay in longer, and if unpleasant effects do not occur, as long as a half an hour, or exceptionally one or even two hours. Nothnagel employs cold baths when the case is one of typhoid in the first two weeks, or even in the commencement of the third, in the case of a strong patient who is not too far reduced, who has no cardiac enfeeblement and generally speaking no complication. He employs lukewarm baths in the second half of the third week, and later still when the patient is weak. When there is cardiac feebleness along with fever, and especially when profuse diarrhœa is present, cold baths should not be given.

Ernest Brand,(a) of Stettin, published an elaborate series of controversial articles in defense of his method of cold-water treatment of typhoid fever. This work is characterized by exuberance of detail and refinement of analysis. The main argument is, however, very simple and in view of the results claimed by the author and established by official reports of the German Government as regards the military service, very important. Brand states that the dissertation owes its origin to articles published within the last few years by Gläser, Ebstein and Senator, attacking the method and claiming equally good results from expectant and modified expectant methods of treatment. The author claims that the expectant method of the present is in no respect different from that of former periods, and aside from the occasional employment of internal antipyretics, its means are the same. Under the expectant method the death-rate according to Liebermeister is 27.3 per cent.; according to Griesenger, 18.9 per cent.; in Kiel, 15.6 per cent.; in Leipsig, 18.5 per cent.; in the Vienna Hospitals, over 20 per cent.;

in Dresden, 13 per cent.; in Strasbourg, 23 per cent.; in Paris, 32 per cent., and during the siege of 1870–71, 60.8 per cent. According to Gläser, the mortality in recent times under an expectant treatment, as well as under the so-called antipyretic treatment, that is to say, with and without medicine, has been reduced to between 7 per cent. and 8 per cent., and quite recently under a purely expectant treatment to 3 per cent.; and according to Ebstein, again, under an expectant management, the mortality has fallen to 5.5 per cent. Brand avers that the favorable statistics of these observers are based upon a concurrence of fortunate circumstances, among which is the unusually favorable characters of the disease in the cases observed. He asserts that what these gentlemen have chosen to designate as the cold-water treatment is in fact not the cold-water treatment as he has practiced and taught it; and he points out the differences between the effects of the cold-water treatment and the treatment itself according to his method and the antipyretic methods in contrasted double columns as follow:—

COLD-WATER TREATMENT. [BRAND'S METHOD.]	THE ANTIPYRETIC METHOD.
1. Severe typhoid will in all its stages be changed to mild; mild typhoid to a still milder form.	1. Changing to an afebrile disease without regard to disturbances of function.
2. That happens: *A*. Through the prevention of any rise of temperature and the control of the temperature within a range scarcely exceeding the normal.	2. *A*. Through the prevention of excessively high temperatures.
B. Through preventing disturbances of normal function.	*B*. Through converting a continued or sub-continued fever to a remittent type.
C. Through limiting the depressing and febrile process.	
D. Through the prevention of complications.	
3. Every third hour a bath, 15° R., fifteen minutes' duration as long as the temperature rises above 39° C. cold affusions, precise and good alimentation.	3. Formerly. Baths when the body temperature rises to 40° C. every second evening, and then some antipyretic drug. At present. In the evening an internal antipyretic, and during the night baths frequently repeated until the temperature falls to normal. During the day no treatment whatever, or at least only when the temperature rises to extreme high levels; then a bath.
4. The treatment is directed against the process as an entirety.	4. Here, only against the symptom of extra high temperature.
5. Baths alone are administered.	5. Baths and internal antipyretics.

6. Mortality: When the treatment is begun at the right time, and carried out according to rule, it will be nil. In family practice, . 1 per cent. In consultation practice, ... 3-4 per cent. In military practice, . 4 per cent. In hospital practice, . 5 per cent. Without great fluctuation.	6. Formerly an average mortality of from 7 per cent. to 8 per cent.; ranging between 0 and 23 per cent. At present, 10 to 18 per cent., with remarkable oscillations.

He claims that Gläser and his followers are wrong in that (*a*) they have confused a faulty antipyretic method with the true cold-water treatment; (*b*) that they have attacked the cold-water treatment and pronounced an unfavorable judgment upon it without having understood it; (*c*) that they ascribe to the expectant treatment results that in fact it does not possess.

He then sets out to give the points of his cold-water treatment, and to show the medical world that it is something entirely different from what the professors of Berlin, Göttingen and Hamburg think; and he quotes from the statistics of the Prussian Army reports from April 1, 1879 to March 31, 1881: "With reference to the treatment of typhoid fever, the cold-water treatment of Brand is very generally used in the army, with the exception of some few small hospitals, whose mortality is not of sufficient importance to modify the general result. The mortality from typhoid fever from 1820 to 1844 was 25.8 per cent. From 1868 to 1874, with the exception of the years of actual field service, 15 per cent."

In the	year	1874	were	treated	2735	cases	with	329	deaths=12	per cent.
"	"	1875	"	"	3620	"	"	408	" =10.9	"
"	"	1876	"	"	2747	"	"	298	" =10.8	"
"	"	1877	"	"	2081	"	"	206	" = 9.8	"
"	"	1878	"	"	2112	"	"	190	" = 8.9	"
"	"	1879	"	"	1741	"	"	163	" = 9.4	"
"	"	1880	"	"	2534	"	"	226	" = 8.9	"

Brand cites cases showing that in the employment of the systematic treatment by cold water, complications need not cause anxiety; that properly administered, it is in heart weakness, as is also especially true in pneumonia, a sovereign remedy; that in pregnancy, in order to save the life of the child, it must be employed from the beginning; that in the severest conditions it affords measures of assistance to the physician not to be found in the shops of the apothecaries; and that neither poverty nor the circumstances even of country life render its employment impossible; and in recapitulation, he formulates the following as the results of the systematic cold-water treatment according to his directions:—

(1) Not only the possibility of preventing every single exacerbation of fever: but also the absolute control of the fever by acting upon its cause. (2) The prevention of disturbance of function on the part of the brain, the heart, the lungs, the kidneys and the skin. (3) The prevention of catarrh of the digestive tract and the possibility of an abundant alimentation. (4) The prevention of the progress of the infiltration of the intestinal glands to ulceration in those cases which are thus treated from the beginning. (5) The prevention of complications in cases thus treated from the beginning, and the lessening of the number of complications in those which are submitted to the treatment at later periods. Altogether, there remains of the ordinary picture of typhoid fever under the Brand cold-water treatment nothing more than (*a*) a mild fever, (*b*) an unimportant bronchial catarrh, (*c*) an enlargement of the spleen, (*d*) the rose rash, (*e*) the infiltration of the intestinal glands. Everything else is prevented, and what might have been the severest case runs its course as a mild one when the patients are sufficiently early brought under treatment. The exceptions to this statement occur only when complications develop at the onset.

Bouchard's[61] treatment of enteric fever comprises four principal points: general antisepsis, intestinal antisepsis, antipyresis and alimentation. As soon as the diagnosis is made, he prescribes (1) a purgative which shall be repeated methodically every third day. For this purpose he uses fifteen grams of sulphate of magnesia. (2) Forty centigrams of calomel daily in twenty doses of two centigrams each,—one every hour for four consecutive days. This medication is intended to meet the indication of general antisepsis. (3) Intestinal antisepsis is obtained by the administration of a magma of 100 grams of powdered vegetable charcoal, 1 gram of iodoform, 5 grams of naphthalin. This is mixed with 200 grams of glycerine and 50 grams of peptone. This mixture forms a black semi-liquid magma which is to be taken in the course of twenty-four hours, in doses of a teaspoonful every two hours in water. Morning and evening the large intestine is washed out by an enema of one part of carbolic acid to one thousand of water. (4) From the first day the patient has eight baths daily until the cure is complete, that is to say until the oscillations of temperature do not extend below 37° C. or above 38° C. (5) Quinine is reserved for those cases in which, despite the baths, the temperature remains high. The doses are 2 grams during the first two weeks, 1½ grams during the third week, and 1 gram during the fourth and fifth weeks. These doses are administered *a coup*, 50 centigrams every half hour. When the full quantity has been given the dose is not repeated until after an interval of 72 hours. The indication for the use of quinine is the rectal temperature of 40° C. in the morning, or of 41° C. in the evening. (6) The aliment consists of soup with barley and given in amounts of from 1½ to 2 litres in the course of 24 hours,—the glycerine and peptones in the above mixture and lemonade with wine.

Grancher[62] gives the following directions for the treatment of typhoid fever in children. In the beginning a purgative may be given and repeated if necessary every third day. Twice a day antiseptic enemata, consisting of 500 grams of water and 5 or 10 grams of borate of sodium. If the tongue becomes dry, a simplified intestinal antisepsis of 2 to 4 grams of salicylate of bismuth associated, if there be constipation, with 2 grams of calcined magnesia, and 1.5 to 2 litres of fluid in the twenty-four hours. Bouillon, barley water, lemonade, milk, and, if there be much prostration, alcohol and extract of bark. For the relief of excessive pulmonary congestion, leeches may be applied to the sides of the thorax or flying blisters suffered to remain only a very short time. In the case of persistent high temperature or excessive agitation, cold sponging or lukewarm baths are to be employed. Cerebral symptoms may be treated by leeches applied to the mastoid region. The most important point of the treatment, however, is the systematic employment of quinine in massive doses, not only as an antipyretic, but as an antiseptic. Grancher entertains the view that the temperature, to some extent at least, is an indication of the intensity of the microbic infection. When the thermometer reaches 39.5° C. or 40° C., or above that, there should be administered, according to the age of the child, 50 centigrams to 2 grams of quinine. The quinine is to be administered at about five or six o'clock in the afternoon in doses of 60 centigrams every half hour in order that the beneficial effects may be experienced during the course of the night. It is remarkable that these doses are followed by sleep. The following day the child awakes with a marked amelioration, sometimes so considerable as to astonish those who behold it.

Dr. Baelz, corresponding editor of the ANNUAL, writes that cold-water treatment was used in 94 cases of typhoid fever and 30 cases of typhus in the Osaka (Japan) hospital for infectious diseases. Result: of the typhoid fever patients, 80 recovered, 14 died; of the 30 typhus patients, 22 recovered, 8 died.

Asta-Buruga[63] reported the treatment of 45 cases of typhoid fever in the Roosevelt Hospital in New York. The treatment was mainly expectant. Equal parts of milk and lime-water constituted the sole diet as long as the fever lasted. Forty grains of subnitrate of bismuth and pepsin, divided into four doses, were given in the milk daily. Whisky was used as a stimulant as occasion required. When convalescent the patients were allowed solid food, commencing with a tenderloin steak, ten or twelve days after the subsidence of the fever, and were generally allowed to sit up about five days later. When the fever was protracted, small doses of Fowler's solution were given, apparently with benefit. Antipyretics were administered in several cases. Antipyrine, antifebrin, the cold pack, the cold bath and the abdominal water coil were used. The mortality was 8.88 per cent.

Vincent[64] treated three pregnant women suffering with typhoid fever with cold baths, after the method of Brand, with the result of preventing abortion and curing all three. Sokoloff[65] treated twenty-three cases of enteric fever by the inhalation of cold air, with favorable results.

Randot[66] having made a careful clinical study of the effects of treatment of typhoid fever with small doses of corrosive sublimate, formulates the following conclusions: (1) Corrosive sublimate is useful in typhoid fever, employed in minute doses, that is to say in daily doses of gram .002 to .005. (2) Administered in an alcoholic potion, it has seemed to diminish the duration and intensity of the febrile process without attendant disadvantages. (3) The prescription of solutions of one-half to one-third the strength required to destroy the bacillus of Eberth, very probably permits us to neutralize in the blood the secondary intoxicants of microbic origin. In other words, this treatment seems to address itself especially to the products of the infectious organisms without being able to destroy or sterilize those organisms themselves. (4) It would be reasonable, in order to obtain a direct concurrent antiseptic action in the intestine, to employ a less soluble microbicide, like napthol for example. (5) This treatment in no way hinders us from meeting by appropriate medication the symptomatic indications which present themselves in the course of the disease.

Haas[67] considers that antipyrine exceeds in usefulness according to his experience all medicaments thus far employed in the treatment of typhoid fever, and especially does it exceed quinine. Its action brings about: (1) Decided reduction of the fever temperature of the body. (2) It controls delirium, and favorably influences all the nervous phenomena of the sickness. (3) There appears to be a favorable diminution of the enlarged spleen coincident with the falling temperature. (4) Unfavorable results were not observed. All the patients entered upon early and favorable convalescence. (5) Only exceptionally was the antipyrine badly borne.

Francis Minot[68] presented observations on the treatment of typhoid fever with antipyrine and thallin, based upon the observation of twenty-four cases in the Massachusetts General Hospital, the object of the study being to ascertain the proper doses of the drugs, the general and specific effects upon the patient, the result from continued doses as compared with its occasional employment, any unfavorable results, and the general effect of antipyrine and thallin upon the course of the disease.

The following conclusions were reached: (1) Both antipyrine and thallin have a remarkable power of reducing the temperature in typhoid fever. (2) In no case was the use of these refrigerants apparently followed by any unfavorable effect upon the course of the disease. (3) The general condition of the patient was more comfortable after taking antipyrine and thallin, which were often followed by sleep. (4) The refrigerant medication by antipyrine and thallin appears to have no specific or decided effect upon the course or issue of typhoid fever. It often contributes much to the patient's comfort, perhaps indirectly promotes his safety. (5) The effect of antipyrine and thallin in promptly lowering the temperature shows that the danger in typhoid fever does not consist in high temperature alone, and that the latter is rather an index of the violence of the abnormal condition which we call fever, though perhaps adding somewhat to the danger. (6) By the internal use of antipyrine and thallin, all the effects which are claimed for the treatment of typhoid fever by the cold bath are readily obtained without the trouble and inconvenience of the lattter method, and without exposing the patient to the dangers of exhaustion and shock consequent on the fatigue of removal from bed. (7) These remedies may be given without danger to the youngest patient in suitable doses; and, indeed, their beneficial effects are more decided and the unfavorable consequences are less observable than with adults.

Fuerbringer[69] and Leyden assert, contrary to the experience of Goetze and Rossbach, that antipyrine has no action whatever upon the course of typhoid fever. The experiments of Fuerbringer go to show that calomel probably neutralizes the toxic products of the bacillus, but in point of fact this action has not as yet been positively demonstrated. Leyden holds the opinion that mercury, properly administered, diminishes to some extent at least the intensity of the action of the virus. Thorner has used calomel with some success. Baginsky regards resorcin, and perhaps calomel also, as possessing some action in modifying the intestinal fermentation. Kaliszcher is one of those who regard calomel as a precious medicament in the early periods of typhoid.

Berezovsky[70] has carried out a series of observations concerning the action of antifebrin in febrile patients, mainly in those suffering from enteric fever. The outcome of his experiments is this: (1) Even small doses—four-grains—produce a marked fall of febrile temperature (0.9° to 2.0° C. in an hour after the administration). (2) The duration of action of the drug is different in different types of fever: it is relatively shorter (1½ to 3 hours) in the case of febris continua; it is longer in remittent fever. The latter may be suppressed comparatively easily; the normal temperature may be kept up by giving four-grain doses every two hours. However, the fever returns immediately after discontinuing the drug. (3) Antifebrin reduces the frequency of the pulse from 8 to 22 beats a minute. (4) The blood-pressure—as measured in the radial artery by means of Basch's sphygmomanometer, an aneroid variety—markedly rises after a dose of antifebrin. The same result is obtained on measuring the arterial tension by means of Filipovitch's palpatometer. (5) The amount of urea decreases with the fall of temperature. (6) Patients take antifebrin readily. The drug markedly improves appetite and does not give rise to any unpleasant symptoms, such as vomiting, etc.

Leclerc[71] found as a result of blood-counting in typhoid patients subjected to treatment by the cold bath, by antipyrin and by acetanilide, that while the cold bath and antipyrin were not followed by deglobulization, the administration of large doses of acetanalide for some time was followed by a notable reduction in the number of red corpuscles.

Kesteven[72] treated a series of cases of typhoid fever with oil of eucalyptus in doses of from five to ten minims. In a disease like typhoid fever where distinct local lesions exist in Peyer's patches, the immediate application of the remedy is, he thinks, practicable. He believes eucalyptus to be a specific remedy and would recommend a very thorough trial of it, not only in cases of typhoid fever, but in all zymotic diseases. Reese[72] relates a satisfactory experience in the treatment of typhoid fever with the phosphates, particularly those of calcium, iron and sodium. These salts are administered together with cinchona alkaloids and aromatics. The treatment is begun with mercurial and saline purging. The purging is repeated every week. Curnow[73] advocates the administration of enormous doses of spirits, twelve to sixteen or twenty ounces or more of brandy in the course of twenty-four hours in connection with wet packing.

TYPHO-MALARIAL FEVER.

Nosology.—Dr. J. Levi,[74] our Corresponding Editor for the Virgin Islands, British West India, states that he has never encountered the hybrid fever to which the term typho-malarial has been applied, and thinks that it is quite time that this compound term should be dropped.

Pathology.—J. Edward Squire[75] read a paper on typho-malarial fever, which term he used to designate a malarial fever which has assumed an adynamic type, such as is presented in enteric fever,—the form he had observed among the troops around Suakim during the campaign of the previous year. Dr. Squire realized that there were two diseases, however, present, both possessing similar symptoms, which led to their being diagnosticated as enteric fever. Post-mortem examination showed the diagnosis to be correct for some of the cases, but in others, similar as to symptoms. The absence of the enteric lesions which were expected to be present, proved that these latter were not in reality cases of enteric fever.

The term typho-malarial fever has been applied to several conditions essentially different. The College of Physicians, following the opinion expressed at the International Medical Congress at Philadelphia in 1876, places the name as a subdivision of enteric fever, and describes it as a combination of malarial and enteric fevers; in other words, a compound fever resulting from the simultaneous action of two distinct poisons. A somewhat similar view was upheld in 1861 before the Epidemiological Society of London by Dr. Russell, who goes so far as to speak of typho-malarial fever as an example of the parallelogram of forces, it being, as he considers, the resultant of the two poisons of malarial and of enteric fever; while Dr. Woodward, who gave us the name, considered it a hybrid between these two diseases. Dr. Squire protests against these views, and gives preference to the opinion that typho-malarial fever is not the result of the typhoid fever poison, but a form of malarial fever; and he defines typho-malarial fever to be "the expression of the malarial poison, or of malarial fever in which intestinal and adynamic symptoms are prominent, causing the illness to simulate enteric fever." The College of Physicians, in saying that typho-malarial fever is a combination of typhoid and malarial fever, probably intends to signify that one is modified by the other, and not that two distinct diseases [a hybrid in fact] are produced, as would be suggested by Dr. Russell's simile of the parallelogram of forces. The belief in hybrid diseases should be passed forever. A specific poison produces a specific disease with certain pathological results; but the symptoms may be modified by a variety of causes within and external to the patient; or two poisons may enter the system together, and one may then delay or modify the manifestations of the other; but no new disease is thereby produced. If the term typho-malarial fever presents nothing to men's minds beyond a modified typhoid enteric fever, a hybrid disease, it is not worthy of a place in our nosology. But if a poison absolutely distinct from that which produces enteric fever and with different pathological manifestations, may under certain conditions cause symptoms closely resembling those of enteric fever, and an illness often mistaken for this disease, then we have a morbid state of much interest and of great importance, and one which has claims to special recognition. This would appear to be the case with regard to the disease now under consideration. The term typho-malarial fever having at length been included in our nomenclature, should remain, and should be transferred from its present position under enteric fever and placed as a subdivision of malarial fever. The pathological signs rather than the symptoms serve to show the difference between typho-malarial and enteric fever, and where the necropsies disclose the Peyerian ulcers of typhoid enteric fever, the latter disease is indicated.

Dr. Squire's experience at Suakim shows that besides cases of true enteric fever, verified by post-mortem, there were other cases diagnosticated as such in which the necropsy showed absence of ulceration in Peyer's patches even after three weeks' illness, and only general congestion of the intestinal mucous membrane.

The course of that form of malarial fever which it has pleased Dr. Squire with others to designate as typho-malarial fever, is thus described: Tonsillitis may occur as a prominent sign. The onset is sometimes more sudden than that of enteric fever and bilious vomiting is often an early and persistent symptom. Diarrhœa is frequent, but not invariable. When present the stools are greenish, or resemble the stools of enteric fever. Congestion may extend the whole length of the alimentary canal, causing a nasal catarrh and symptoms resembling dysentery. Later on, the tongue becomes dry and brown and sordes appear. Mental apathy gives place to low, muttering delirium with subsultus and other signs of the typhoid condition, and death may result from exhaustion; or prolonged convalescence keeps the patient in the hospital for weeks or months before the absence of diarrhœa and evening fever allows of his discharge.

The temperature, although often resembling that of enteric fever, reaches a high point earlier in the illness, and the daily range is greater. The absence of rose-spots is invariable. The symptoms being so similar to those of enteric fever, we must turn to the pathology to show the great distinction between the two, their differentiation being of importance from the differences in etiology. Speaking generally, the difference is that whereas cell proliferation and subsequent ulceration in Peyer's patches and the solitary glands of the ileum is the pathological sign of enteric fever, such ulceration is rarely, if ever, found after death from typho-malarial fever. As in other malarial fevers, ulceration may be found in the intestines in typho-malaria, but it does not select and is not confined to Peyer's patches and the solitary follicles. These ulcers may be found in any part of the alimentary canal, and may be of any shape and of almost any size. The other pathological signs are those of malarial fevers: congestion and ecchymosis of the intestinal mucous membrane, especially in the duodenum and upper jejunum, enlargement of the spleen and of the mesenteric glands, and congestion of the liver. Among the complications met with, hæmorrhage from the bowels, urinary organs, and other parts is not uncommon. Pulmonary congestion or pneumonia may occur. Jaundice may be present. Purpuric blotches, without scurvy, were seen in some of the cases at Suakim. Rheumatism, sometimes with effusion into the joints, has been observed. Typho-malarial fever may occur wherever malaria is found; imperfect sanitation or the prevalence of enteric fever may determine the intestinal symptoms. Overfatigue and excitement are predisposing causes. Typho-malarial fever is not communicable from person to person. It is probable that the fever of Gibraltar, Malta, and the Mediterranean may sometimes be of the nature of typho-malarial fever. Dr. Squire proposes that the term should be restricted to malarial fevers which in their symptoms closely simulate enteric fever, and that those cases should be excluded which are found post-mortem to have the pathological appearances of enteric fever.

J. W. Penn,[76] having encountered numerous cases of typhlitis and perityphlitis accompanied by a protracted form of fever presenting many of the characteristics of so-called typho-malarial fever, was led to conclude that a milder degree of irritation in that locality might give rise to a symptomatic fever corresponding to so-called typho-malarial fever.

THE MALARIAL FEVERS.

Etiology.—Osler[77] gave an account of the hæmatozoa which have been found in persons suffering from the various forms of malaria. Knowledge of the blood changes described dates from the researches of Laveran in Algiers which were communicated to the Paris Academy of Medicine in 1881–82 and were finally embodied in a large work on malarial fever published in 1884. Laveran found as characteristic elements in the blood of persons suffering with malaria: (1) crescentic pigmented bodies; (2) pigmented bodies in the interior of the red corpuscles which underwent changes in form described as amœboid; (3) a pigmented flagellate organism. These forms he regarded as phases in the development of an infusorial organism which he considered to be the germ of the disease. These observations were confirmed by Richard, Marchiafava and Celli [who gave to the organism in question the name of plasmodium malariæ], and by Councilman, of Baltimore.

Osler describes in detail and at considerable length the various forms assumed by these bodies, as (1) Amœboid bodies in the red corpuscles. (2) Pigmented bodies in the red corpuscles. (3) Larger solid bodies in the interior of vacuoles. (4) Free pigmented crescents, which crescents may sometimes be seen to develop in the interior of the red corpuscles. (5) Rosette forms. (6) Scattered small bodies, the result of segmentation of the rosette forms,—also described with great fullness by Golgi. (7) Flagellate organisms, round or ovoid or pear-shaped, with finely granular protoplasm containing pigment with flagella variable in number, one, three or four being observed at different times. (8) Small round pigmented bodies one-fourth to one-half the size of the red corpuscles.

The relation of the various forms was not fully established. Both the amœboid and pigmented bodies were met with in both acute and chronic cases, appearing, however, to be more especially characteristic of the acute manifestations of the disease. The hyaline non-pigmented forms and vacuoles containing solid bodies were also more common in the acute cases. The crescents appear to be associated with the more chronic forms of malaria and acute cases which have been under treatment for some time. The rosette form, with its peculiar rosette segmentation, occurred in six cases, invariably in association with the amœboid intra-cellular bodies. The flagellate organisms were present in seven cases, six of which were chronic cases and one an acute case of three weeks' duration. The small free pigmented bodies were more abundant in the chronic form with cachexia. In regard to the relation of these forms to the paroxysms, there were instances in which the amœboid organisms were decidedly more numerous before and during the paroxysm than in the intervals. There were others in which the number during the chill and hot stage was so small that examples were very hard to find. In others again, slides taken before the attack and during each stage were negative, and yet in subsequent paroxysms the bodies were present in the blood. Osler holds the opinion, all things considered, that the pigmented bodies on the red corpuscles are more numerous before and during the attack, but the difference is by no means striking. The segmentation seems in some way associated with the paroxysm, but its relationship requires further investigation. As to the influence of medicines upon the organism, quinine invariably causes the pigmented bodies to disappear. In recent cases this remedy acts as a positive specific against these organisms just as it does against the malady itself. Arsenic does not seem to influence the pigmented intracellular bodies. Thallin and antifebrin had no effect.

Until the true affinities of the organisms are determined by an expert, its proper place seems to be the genus hæmatomonas of Mitrophanow, which conveniently includes all monads parasitic in the blood. Thus, genus, hæmatomonas; species, hæmatomonas malariæ; definition, body plastic, ovoid, globose, no differentiation of protoplasm, which contains pigment grains. Flagella variable from one to four, highly polymorphic, occurring in (1) amœboid form; (2) crescentic encysted form; (3) sporocysts; (4) circular free pigmented bodies. The name designates the natural affinities of the parasite, its habitat and the conditions under which it grows; on which grounds it seems preferable to that of plasmodium malariæ suggested by Marchiafava and Celli.

Councilman[78] announced at a meeting of the Pathological Society of Philadelphia in October, that he had found in greater or less abundance the whole series of organisms described by various observers as existing in the blood of malarial patients. The segmental organism he found in a large number of cases just before and during the chill, and believes it to be characteristic of that stage. The crescentic organism he found solely in malarial cachexia, in which condition, when intense, it often exists in extraordinary abundance. Blood taken directly from the spleen was found to be especially the home of the various organisms, and it was chiefly from it that the flagellate forms were obtained. He had carefully studied the effect of the administration of quinine upon these organisms, and found its influence, in large doses, upon the form which accompanies the acute intermittent paroxysm and which is characteristic of the chill period, most pronounced. The administration of fifteen grains of quinine upon three or four successive days is often followed by the almost complete disappearance of the organism, whilst the administration of forty-five grains daily for three days, always causes the organism to completely disappear. On the other hand, the crescentic organism of malarial cachexia does not appear to be particularly affected by quinine.

E. Maurel[79] announced to the French Association for the Advancement of Science that it is always easy to distinguish a healthy from a malarial soil. The water taken from malarious districts always contains numerous micro-organisms, some of which are possibly Laveran's corpuscles in an early stage of their development. In regard to the real value of Laveran's corpuscles in the production of malaria, he believes them to be indirectly concerned in the production of the affection, although their relation to it has not yet been absolutely demonstrated.

Symptomatology.—Page[80] found in more than two thousand cases of sick children observed among the poor of New York, about five per cent. to be suffering from malaria. The chief symptoms are anorexia, more or less frequently constipation, coated tongue, sweating and pyrexia. Anæmia of a high grade is common. Only four or five per cent. of the cases were jaundiced. Twenty per cent. of the cases had bronchitis. The treatment was commenced by the administration of small doses of calomel followed by a saline, and this followed by quinine during the continuance of the fever. Sometimes the quinine was administered in anticipation of the paroxysm. In the stage following the fever, Fowler's solution proved itself superior to quinine, and the subsequent anæmia was most efficaciously treated by the tincture of the chloride of iron. Nux vomica and bitter tinctures proved good after tonics. These cases yielded to treatment better than similar cases in adults.

Singer[81] reported a case of acute polyneuritis following a grave attack of malaria which occurred in Singapore.

Treatment.—According to Rouquette,[82] the malarial microbe gives rise to symptomatic fever by reason of its activity in producing leucomaines. During the access of fever the microbe is eliminated by the natural emunctories. The liver is a great destroyer of leucomaines. If an inhabitant of Algiers who has never had an access of fever, develops ague on returning to France, it is for the reason that his liver has to some extent lost its power of elimination and that it does not sufficiently destroy leucomaines, it being, according to this author, due to the fact that the liver and spleen are organs of elimination that these organs undergo hypertrophy. According to Rouquette, the primary indication in malaria is to restore these organs to their normal volume. To accomplish this purpose, he injects directly into the tissue of the spleen a Pravaz syringeful of the following solution: aqueous extract of ergot [Bonjean], 2 grams; water, 10 grams; glycerine, 10 grams. This operation is repeated at intervals of some days, once or twice.

Alberto Solaro,[83] at the suggestion of Professor Fenoglio, treated five cases of malarial splenic tumor by the injection of Bonjean's ergotine diluted with water, in doses of from .05 to .35 grams. He summarizes his results as follow: (1) The injection of ergotine into the spleen is useful in bringing about resolution of malarial enlargement of that organ. (2) The general condition is improved. (3) The injection is followed by rise of temperature. Query: Is this rise caused by the malarial infecting principle previously latent in the spleen finding its way into the general circulation? (4) It is necessary to use quinine in connection with the injections of ergotine. (5) This treatment should not be employed in individuals extremely reduced.

Savitzka[84] found that a combination of ergotin with quinine acts very satisfactorily in intermittent fever, especially when accompanied by an enlarged and tender spleen, and that in this way a considerable quantity of quinine can be saved, as one-half the dose of quinine which would be required if given alone, will suffice if combined with ergotin. The preparation of ergotin used was Bonjean's, the dose in chronic cases being about one grain three times a day.

Soshinski has been in the habit of prescribing phosphorus in all of his cases of intermittent fever during the past five years and has had excellent results from its use. He administers five drops of the oil of phosphorus well diluted, three times a day.

H. M. Clark[85] treated no less than ten thousand cases of malarial disease with picrate of ammonium and kept a record of half of these cases. In nine cases only out of the five thousand did the remedy fail. The usual dose is from one-eighth to one and one-half grains, four or five times a day, in pill form. One-half a grain is the average dose. All forms of malarial disease yield promptly. Picrate of ammonium does not produce headache, deafness, tinnitus, etc., nor does it disorder digestion or cause nausea. His experience has led him to the conclusion that in all varieties of intermittent fever, and in malarial neuralgias, picrate of ammonium is a valuable antiperiodic, and it is an efficient and perfect substitute for quinia. It has the following advantages over quinine: (1) It is much less expensive. This is an important consideration where hundreds of cases of malarial diseases have to be treated annually. (2) The dose given is very much smaller. (3) It does not produce the unpleasant effects that quinine does,—headache, deafness, tinnitus, etc.; nor does it disorder the digestion or cause nausea, as quinine is apt to do, in the doses in which it has sometimes to be given.

Dr. Joseph Levi, Corresponding Editor of the ANNUAL in the Virgin Islands, has used the permanganate of potassium in the treatment of chronic malarial affections with very interesting and successful results. The usual dose is one-half to one grain in water three times a day. He likewise affirms that all chronic affections due to malarial toxæmia have been treated with great success by this drug. Putauski[87] recommends deep hypodermic injections of muriate of quinine in cases of intermittent fever that do not yield to the internal use of the drug. A mixture of one part of the salt in two parts of water is injected hot. From two to three grains of the salt are administered at a time. The injection is said not to be painful. It neither irritates the tissues nor causes induration. Riggs[88] treated hæmorrhagic malarial fever without quinine, but with large and frequently repeated doses of arsenious acid,—from one-tenth to one-fortieth of a grain at intervals of three or four hours until one-half or three-fourths of a grain are taken. The symptoms subside within twelve hours. Upon convalescence, quinine, iron, arsenic, and nux vomica were administered. Caution is advised, lest gastric irritation or arsenical poisoning result.

Antony[89] successfully treated the continued forms of malarial fever, both at the beginning of the indisposition and in the cachectic period, with antipyrin, when quinine was manifestly without effect. He found it also to be an excellent auxiliary to quinine.

Luzzatto[90] treated malarial fever by carbolic acid, resorcin and iodoform. Gram 0.5 of carbolic acid was given daily in divided doses to a male 17 years of age. A remarkable amelioration of the disease, attended by a great reduction of the temperature, at once took place. On the sixth day of the treatment by carbolic acid, the tenth of the disease, the temperature ceased to rise. The cure was lasting, as no relapse occurred. The rigor was much shortened and the free intervals became correspondingly longer. Two other cases were treated in the same way with similarly favorable results. In the cases treated by resorcin, the remedy was administered subcutaneously in three doses of gram 2.20. The action was almost identical with that of carbolic acid, and the author looks upon the two as analogous in their therapeutic effects. Iodoform shortened the rigor and moderated the temperature, but did not completely suppress the attack, nor did it in all cases always prevent relapse after the medicine was discontinued. In four cases treated, complete recovery took place under its administration. For the treatment of the splenic tumor resorcin and ergotin were injected together, with the gratifying result that a decided reduction took place in the size of the organ.

MOUNTAIN FEVER.

Curtin[91] contributed to the transactions of the meeting of the American Climatological Association for 1886 an interesting and important study of the so-called Rocky Mountain fever. He quotes Dougan as saying that the term "mountain fever" has by long continued and frequent use almost established itself in the nomenclature of disease in the mountain districts of the West. Whether or not its use is proper as designating a separate and distinct type of fever, a pathological entity, may well be questioned. Dr. Curtin believes that proof of this is wanting.

Van Eman[92] thinks that all of the Western fevers belong to one of three classes: (1) febricula, or simple irritative fever; (2) malarial, which may be divided into two general subclasses, remittent and intermittent; (3) typhoid, to which class belong not only the well-marked typical cases of typhoid, but also nearly all of those now usually called typho-malarial,—"a name for which there is no excuse."

TYPHUS.

Kiernan[93] discusses errors of diagnosis which occurred during a local epidemic of typhus. The patients were tramps, and in the main much debilitated. In several cases the mental condition was the chief source of error. Among the conditions mistaken for typhus were delirium tremens, acute alcoholism, sunstroke, pneumonia, chronic nephritis complicated by malaria, and accompanied by delirium, sub-acute gastritis, cerebro-spinal sclerosis complicated by malaria and erythema, cerebro-spinal fever, malaria, phthisis and pleurisy complicated by delirium, small-pox, scarlatina, pyelitis, diarrhœa, dysentery, diphtheritic dysentery, scrofula, rheumatism and pharyngitis.

MILIARY FEVER.

Brouardel[94] reported to the Paris Academy of Medicine the principal facts in regard to the epidemic of miliary sweating which prevailed in the departments of Sienne, Haute-Sienne and Indre. There is no doubt that this disease was idiopathic miliary fever, the historical sweating disease. Daremberg[95] wrote an important letter on the subject to the *Journal des Débats*, ably translated by the *British Medical Journal.*

Daremberg accompanied Drs. Chantemesse and Thoinot to Montmorillon,—the centre of a district where more than 4000 cases, with 300 deaths, occurred. The disease is characterized by a profuse sweat and an eruption of small miliary vesicles. M. Littré believed that the disease was known to Galen, but the first authentic epidemic was the *sudor Anglicus*, "sweating-sickness," or *suette Anglaise* of 1485, well known to historians. It was very fatal, according to Delaune, in London in that year, and Stow records that in 1499 it raged in the metropolis, so that Henry VII. and his court removed to Calais. In 1506 to 1507 the epidemic was terrible: Oxford was depopulated. In 1529 an epidemic ravaged Europe. In the seventeenth century it appeared in France and Germany. The mortality was less, but the miliary rash appeared in every case, whilst in the first epidemic it was rare. But Dr. Daremberg shows that the patients in the English epidemic of 1485 mostly died within a few hours of the appearance of the first sweat, before the rash had time to develop. Precisely the same thing has occurred this year, several patients having died in two or three hours without any trace of rash. In the same district very mild cases have occurred, the patients being confined to their beds for a couple of days and then able to work. As in an epidemic in Poitou. in 1845, and another in the Aude, in 1864, the epidemic was preceded by many cases of eruptive diseases mistaken for measles. The troops of the garrison of Poitiers suffered severely from this "scarlatiniform measles." The first case of sweating-sickness in 1887 occurred on April 16 in the commune of Saulgé. In the town of Montmorillon (5128 inhabitants) 47 persons died of the epidemic between May 3 and June 19. At Moulismes (940 inhabitants) there have been 45 cases and 16 deaths,—the mayor, two other officials, and the wife of one of those magnates dying within a few hours. At St. Remy (893 inhabitants) there have been over 50 cases. In five or six communes hardly a dozen people have escaped infection. The more recent cases have been mild. Dr. Daremberg found that the patients had little pyrexia, dyspnœa, or palpitations. They had profuse perspiration and an eruption, in some cases resembling measles, in others scarlatina, but always vesicular. The vesicles on the tongue formed ulcers. The patients felt no prostration and little loss of appetite. The earlier cases were of quite a different type. The patients showed the worst symptoms of malignant fever, profound depression, violent delirium, syncope, epistaxis and intestinal hæmorrhage. A great number of intermediate forms proved the identity of the very bad and the very mild cases. The alleged anomalous forms of measles seen in the same district are also forms of sweating-sickness. Drs. Brouardel, Chantemesse, Descout and Thoinot have inspected the infected communes, and enforced, through the sanitary authorities, disinfection by means of sulphurous and carbolic acid. The peasants at first objected to these measures, and dreaded the damage which the acids might inflict on their furniture. They preferred the common French placebo, infusion of lime-tree blossoms, to quinine, and when stricken they closed their windows and piled eiderdown quilting and blankets over their bodies, notwithstanding the intense heat of the weather and the fœtor developed by the practice. Dr. Daremberg believes that the disease is infectious. Another medical correspondent, signing himself "Dr. G. D.," writes to the *Journal des Débats*, noting the fœtid perspirations, the measles-like variety, and the extreme infectiousness of the disease. Some recent cases of so-called measles at Bourges appear to him to be sweating-sickness.

Parmentier,[96] who accompanied the commission sent out under the direction of Prof. Brouardel to investigate this outbreak, has published a long account of its clinical features and epidemic relations.

CHRONIC ALTERNATING PYREXIA IN PSEUDO-LEUKÆMIA.

Ebstein[97] read a paper entitled "A Case of Chronic Recurrent Fever; a New Infectious Disease." The illness began in the patient, a male, aged 19 years, without distinct cause and the time of the onset was not clearly marked. The only symptom of the disease was the fever. The patient had feverish attacks which ceased after a time and recurred after a definite interval, so that the pyrexia and apyrexia alternated with a regularity not seen in any other disease. He had nine attacks of this kind between October 13th, 1886, and July 11th, 1887,—that is in a period of 211 days. At the time of the reading of the paper he was suffering with the tenth attack, then not yet completed. This last attack was preceded by an interval of apyrexia lasting thirteen days. The temperature was recorded only from November, 1886; but it is probable that in the previous September and October he had already had two attacks. Each of these lasted, as a rule, thirteen to fourteen days, being succeeded by a period of apyrexia of ten or eleven days' duration; so that from the height of one attack to the corresponding period in the next attack the interval was about twenty-four days. In each attack the temperature rose gradually to 40° or 41° C. or even higher, and then gradually fell to subnormal ranges. The lowest temperatures recorded were 35.6° and 36° C. When the morning temperature reached the normal, the evening temperature was still slightly febrile. With one exception, probably due to the occurrence of a brief attack of intercurrent pleurisy, the course of the temperature was absolutely uniform. The patient's intelligence remained clear, there was some weakness of the heart, the blood was normal, showing neither increase in the number of white corpuscles nor bacteria. The lungs and digestive organs were quite healthy. There was neither sugar nor albumen in the urine. The spleen was enlarged. Treatment directed against the fever and consisting of arsenious acid, quinine and antipyrin, with brandy as a cardiac stimulant, had no effect. About July 7th the fever was almost continuous, and death occurred with general œdema and coma on August 14th. The autopsy revealed the lesions of lymphadenoma of the bronchial, mediastinal and retroperitoneal groups, with growths in the lungs, pleura and liver. The case was in fact one of Hodgkin's disease, or pseudo-leukæmia with an unusual type of pyrexia, which however had been previously described by Murchison, Gowers and Pel. In no other instance on record was the alternating pyrexia so distinctly characterized. A writer in the *Medical News* particularly criticises Ebstein's view that the case is to be regarded as a new infectious disease, and also his title of chronic relapsing fever.

Pel[98] replied to Ebstein regretting that the latter had taken no notice of two other cases which he had published in 1886, which closely resembled Ebstein's case. Pel does not think the disease entitled to a new name. He calls it an extremely infectious form of pseudo-leukæmia.

HECTIC FEVER.

Pribram[99] treated hectic fever with ounce doses of a one per cent. solution of antipyrin, followed by half ounce doses of the same solution every hour until the temperature fell to normal. Two doses usually proved sufficient. If the one per cent. solution seems strong enough, it should be continued in the same manner for four or five days. If not, a one and a half or even a two per cent. solution should be used. On the sixth or seventh day omit the second dose. If three doses have been given daily, omit the third. After a few days longer, drop the second. He claims excellent results.

THERMIC FEVER.

Ellsbury[100] reported the case of a patient who, after violent exertion during a heated term, became delirious and maniacal. The case seemed to be one of thermic fever with the peculiarity that the pulse rate and the temperature were normal. Lewis[101] suggested the administration of ten grains of antipyrine hypodermically in these cases.

YELLOW FEVER.

Freire[102] completes a communication recently made concerning the prophylaxis of yellow fever, by submitting a statistical table by which it appears that 6542 persons have recently been inoculated in Rio Janero with attenuated virus, and that of this number eight only subsequently contracted the fever, although a very serious epidemic was then prevailing. In contrast with this mortality of about one in a thousand of inoculated persons, there was a mortality of one in one hundred in persons not subjected to the treatment; consequently, according to the author's conclusion, nine out of ten were saved by the procedure.

Finlay[103] contributed a paper giving the results of his bacteriological studies in yellow fever. He made, in connection with Delgade, numerous cultivations with the blood and secretions in agar-agar and broth. Orange-yellow and milk-white colonies of micrococci developed, either separately or together in the course of the inoculating needle, forming about the puncture on the surface of the jelly a salient disk with fringed edges. The yellow colonies consisted of micrococci endowed with active movement, arranged singly and in groups of two or three. The micrococci of the white colonies were rather larger, elliptical in form and of less rapid movement. These colonies were easily cultivated separately.

Lacerda[104] studied the bacteria found in the bodies of persons dying of yellow fever, and made cultures of the bacteria taken from parts of the tissue and the blood. He found bacteria in the form of small chains, formed by a series of granulations having nearly equal dimensions, an outline somewhat elongated, and approaching the cylindrical. Characteristic of these bacteria, in contrast with all others, was the tendency to constantly present a ramifying arrangement.

De Arenas[105] reported yellow fever in a monkey. The symptoms were well-marked and the case terminated fatally.

Hebersmith[106] treated cases of yellow fever with injections of pilocarpin muriat, in quarter of a grain doses, with very favorable and prompt results. The series of cases was, however, limited.

VARIOLA.

Symptomatology.—Cotugno,[107] in a recent epidemic of small-pox at Naples, encountered convulsions in all forms of the disease, and not only in the graver forms, as certain authors have pretended is the rule. The occurrence of convulsions is no criterion for prognosis; but the irintensity, extent, duration and frequency of attack furnish grounds for reasonable prognosis.

Montefusco[108] made studies in local thermometry in small-pox, which resulted in the discovery that in the splenic region there is a constant increase of temperature relatively as compared with that of the hepatic region and the abdominal walls at large. There is no constant relation between the local temperature of the spleen and the axillary temperature.

Prevalence.—Dr. Beshara Manasseh, Corresponding Editor in Syria, writes that small-pox prevailed very extensively there during the year, among all classes of society, and was very fatal. Dr. Neve, Corresponding Editor in Kashmire, India, states that it also prevailed in Kashmire and surrounding districts, the death-rate being not less than 49 per cent.

Treatment.—Œtrös[109] treated 315 cases of severe small-pox with xylol and obtained excellent results. Vinay and Riche[110] are partisans of the cold baths during the period of invasion and eruption. In the period of suppuration tepid baths are to be preferred. Colleville[111] recommends a vaseline ointment of iodoform of the strength of one to twenty, not only as an antiseptic emollient grateful to the patient, but as somewhat anæsthetic and as preventive of the formation of foul-smelling scabs and of pitting.

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DISEASES OF THE MOUTH, STOMACH, PANCREAS AND LIVER.

By W. H. THOMSON, M.D.,

NEW YORK.

DISEASES OF THE MOUTH.

APHTHOUS AND ULCERATIVE STOMATITIS.

Oidium Albicans.—As a cause of obstinate stomatitis this fungus has long been recognized. But the conditions, local or constitutional, which favor its development are still obscure; for some of the most inveterate cases occur in persons otherwise healthy, though most commonly there are present concomitant serious states of malnutrition. Ch. Audry[1] contributes an interesting article on the remarkable polymorphism of this fungus according to the media, solid or liquid, which may be chosen for its artificial propagation. On plate gelatine (neutral or slightly alkaline, or acidified with phosphoric acid), also on agar-agar, with or without addition of glycerine, and on potato, the growth, originally derived from aphthæ in the mouth of a woman with peritonitis, was in each quite similar, consisting of colonies of monocellular forms, generally round or oval, sometimes irregular, and reacting to color tests strikingly like spores. In liquid media the results were very different. In bouillon the round bodies assumed an elongated shape, and then in many cases threw out long, clear filaments, which developed into striated tubes ending in flask-shaped corpuscles, without, however, their ever containing any thing like spores. In sterilized wine true mycelial tufts and growths developed, without, however, the filaments observed in bouillon; so that it did not coincide with the well-known mycoderma vini. While this growth proceeded, no acid odor developed in the wine; while the bouillon, on the other hand, did become highly acid. On transplanting this altered growth from either the bouillon or the wine back to the solid media, indifferently gelatine, agar-agar or potato, they soon lost their filaments, the elongated forms disappeared, and the growth reverted to the condition of monocellular bodies above described. He mentions that the addition of a little borax wholly prevented their development in bouillon.

Dr. B. M. Ricketts[2] mentions a case of a gentleman who presented ulcers covered with the white patches of this affection on the hard palate, apex and base of the tongue, which resisted applications of glycerine tannate, corrosive sublimate solution (1–500) and argent. nit. gr. v to ℥j, and which yielded at last only to the solid stick of the nitrate of silver applied thoroughly to the ulcers every other day. Sevestre[3] advises iodoform incorporated in lard for children.

Professor Bouchard[4] goes quite thoroughly into the many varieties of stomatitis with and without membranous exudations, and draws a distinction between the ulcerative and the non-ulcerative or benign forms. The latter may be both discrete and confluent. The confluent form may be confounded with the more serious ulcerative disease, as it extends in broad patches sometimes on the inner aspect of cheeks and pharynx, accompanied by desquamation of the tongue. It differs from ulcerative stomatitis in that the latter does not affect the pharynx, while, moreover, its patches are quite superficial and can be easily detached from the underlying membrane, are never offensive, have no fever or severe constitutional symptoms present, and the affection is never contagious. There is much local distress in marked cases, however, and difficulty of swallowing; and in the confluent form the trouble may persist for a month. Bouchard insists upon the affection being of a herpetic character from the beginning, and therefore being very prone to relapse. The best treatment is by using, a number of times a day, especially after meals, a concentrated solution of salicylate of soda as a mouth wash.

Fournier[4] describes a recurrent buccal herpes which occurs in adults who may be suspected of syphilis, and who, indeed, may have had that disease. But that the affection is not syphilitic is proven by its recurrence and disappearance without any relation to syphilitic treatment, and hence should not be regarded as such so as to lead to the prohibition of marriage.

Dr. Hiram Corson,[5] of Conshohocken, Pa., speaks of a long experience with the infusion of the inner bark of the root of the common sumach (rhus glabrum) as the best of remedies in all forms of stomatitis, mercurial ptyalism, cancrum oris, etc. The bark is placed in a bowl and boiling water added, when it soon makes a mucilage, which, when used as a mouth wash, affords great relief to the soreness and pain and speedily arrests any ulcerative process. This remedy is also recommended by Dr. H. R. Wharton[5] as a wash, with chlorate of potash. Seven cases of gangrenous stomatitis were brought from an orphanage in Philadelphia, in the month of July, to the Children's Hospital. The affection had developed during an epidemic of measles. The tendency of this exanthem to cause gangrene the editor has seen illustrated in two cases of death from gangrenous sores in the chest, following the ignorant application by the parents of mustard poultices to allay the cough. Dr. Wharton's treatment was to etherize the patients, then remove the loosened teeth, scrape the sloughing tissue away, and swab the whole of the gangrenous surfaces with fuming nitric acid; after which the mouth wash was frequently employed. Two of the cases died with the usual antecedents of diarrhœa and pneumonia and the others recovered. The editor's own experience leads him to prefer the application of Smith's solution of bromine, undiluted, as more effective against all forms of gangrene, including diphtheritic sloughs, than any other local application, and as more likely to prevent further development of the process in contiguous parts.

The liability to pneumonia in all forms of serious stomatitis (vide Descroizeilles[4]) is a fact of clinical interest, and especially in connection with the claim of Sternberg,[6]—that Friedländer's pneumococcus is not the agent which causes croupous pneumonia, but that it is due to a micrococcus discovered by him in normal saliva, and to which he has given the title of micrococcus-pasteurii. This micrococcus is likewise regarded as holding a greater relation to pneumonia by Fränkel and Weichselbaum. How its spread from the mouth to the lungs, so as to excite pneumonitis, occurs, is as yet unknown.

DISEASES OF THE TONGUE.

Tuberculosis.—Delavan[7] discusses lingual and buccal tuberculosis, of which he reports 7 cases. Of 21 recorded cases of lingual tuberculosis only 2 were females. The disease was primary in no less than 9 out of the 26, secondary to disease in the lungs or larynx in 9, and not stated in 8 cases. Its most common location was at the tip of the tongue, sometimes at the side and dorsum, and in only 1 was it at the base of the tongue. It may occur at any age, for the primary cases ranged between 11 and 71 years of age. The prognosis is always grave, and the disease runs its course with comparative rapidity,—the longest of the primary being two and a half years, and the shortest ten weeks. In the primary cases the diagnosis from epithelioma may be difficult, and not certain without the use of the microscope; while in the stage of tumor before ulceration they may be readily taken for syphilitic gummata. Dysphagia soon develops, and toward the close becomes very severe.

The treatment should be radical in all the primary cases, with the hope not only of relieving the local symptoms, but of preventing constitutional infection. Dr. H. T. Butlin[21] recommends extirpation of the diseased parts, and, if necessary, excision of the tongue itself. Where the ulcerations, however, are neither extensive nor deep, Dr. Delavan recommends touching them with lactic acid; and if this does not afford good results, then the application of the galvano-cautery.

Fissures, etc.—Dr. Butlin recommends that ulcers, fissures and vesicles of the tongue should be touched with 1–2 per cent. sol. chromic acid. He objects to the silver nitrate in these, as also in smokers' spots, leucoma and ichthyosis, which admit of palliative treatment only and not with caustics, owing to their liability to change into cancer,—to which disease he also considers alcohol, smoking, and the eating of raw meat as predisposing. For such conditions his most successful treatment was by the application of one part of salicylic acid to ten parts each of alcohol and water three times a day. Sclerotic glossitis indicates tertiary syphilis, and is the precursor of the deep fissures and puckerings seen in syphilitic tongues. Hemiglossitis is probably of neurotic origin in most cases, in others is a milder form of general acute glossitis.

Black Tongue.—The curious affection named black tongue is not so uncommon as supposed. The editor himself has had two well-marked cases,—one of them in a physician who, after many years of a coated tongue from dyspepsia, had his mouth feel one day as if it were filled with hairs, and, on looking at his tongue with a glass, was surprised to find the posterior two-thirds colored quite black, and with long filaments proceeding from the swollen papillæ. This condition continued for over a month, and resisted a good many local applications, when it ceased soon after he left off taking strong English cheese for lunch. Butlin regards black tongue as of a parasitic nature, and probably dependent upon alterations in the normal micrococci present in the mouth forming the major part of the coating. The amount of micrococci is in inverse proportion to the epithelium and food present. The bacilli adhere mostly to the papillæ filiformes, and hence the clean tongue of little children in whom these papillæ are not well developed.

Roth[9] reports two cases of this affection with hair-like projections,—both in apparently healthy men. In the first, the tongue gave an acid reaction, and the breath had an acid odor. The microscope showed the coating of the tongue to be composed of various fungous growths with epithelial cells,—the hair-like projections consisting of epithelial cells piled upon each other, with colonies of micrococci between them. Under repeated applications of a 10 per cent. solution of salicylic acid every three or four days, the coating gradually disappeared. In the second case the patient had suffered from fetid breath for ten years. There was no nasal or pharyngeal disease nor carious teeth; and Dr. Roth found, by scraping some of the coating off, that the bad odor was from it. Salicylic acid seemed of benefit to the tongue, but did not affect the odor; whereupon a 10 per cent. solution of corrosive sublimate was applied, and after five applications the patient was apparently cured, the tongue now giving a neutral reaction and only very few micrococci.

C. Böcker,[10] of Denmark, describes, in almost identical terms with Roth, the microscopic appearances in two cases of black tongue in two healthy country-women, aged between fifty and seventy. Scheet,[11] however, denies that this affection is a mycosis, and maintains that it is a pigmented hypertrophy of the papillæ, which he finds are avoided by the ordinary micrococci of the mouth. In this he disagrees with most authors, who, after Henois, have found the association of these growths with micro-organisms constant, and the treatment based on this assumption the only effective one. Roth's case, above cited, is interesting as affording another possible source of bad breath.

New Disease of the Lips and Mouth.—Mr. Jonathan Hutchinson has recently drawn attention to a form of inflammation of the lips and mouth, in which superficial ulcerations occur, followed sooner or later by some form of skin disease, and leading to a fatal termination. The exact form of skin disease varies, but the hands and feet are the parts usually affected, the nails being especially prone to suffer. In some cases the eruption may consist of bullæ, followed by free papillary outgrowths. No special antecedents can be alleged as the probable cause of this malady, but the disease has only been observed in middle life or in early senile periods. If not checked by treatment the disease appeared to run its course in about six months, death being produced by exhaustion. Mr. Hutchinson considers that opium, given in repeated doses, will cure it, and that there is hardly any tendency to relapse afterward. All the best marked cases he had observed in males, but several milder were women. Careful inquiry had failed to support the suspicion that the disease might be due to contagion from animals. He has lost two patients by death. Both were those first observed, and since the discovery of the signal efficacy of opium no case has ended fatally. The observation as to the efficacy of opium was made simultaneously by Mr. Pollock and Mr. Hutchinson, two different patients being at the same time under their separate treatment. Since that every case yielded if the dose of opium were sufficiently pushed. In one, however, the disease did not yield quickly, and for more than a month seemed likely to end in death. As regards permanency of cure, two patients had remained quite well four years after their recovery, and in a third a period of two years had elapsed. One thing particularly noted was that no case had indicated any tendency to spontaneous improvement. In all cases the inflammation of the mouth and lips took precedence of the skin symptoms, and in some the latter were very slight, while not one of the patients belonged to the poorer classes. Hutchinson thinks the disease allied to other forms of skin diseases occurring in early senile periods with disturbed health, such as certain kinds of pemphigus, pityriasis rubræ, psoriasis, and lichen planus. In confirmation of this suggestion he mentions the case of an elderly lady who had a kind of spreading eczema,—psoriasis of the hands and scalp. She lost her hair and nails, and was rapidly failing in health, when she recovered quickly and completely under opium.[12]

DISEASES OF THE STOMACH.

DILATATION.

Many articles by American and European writers still appear on stomach dilatation occurring without pyloric stenosis as a cause. There can be no doubt that a veritable pathological condition of this kind often exists as a cause of serious impairment of digestion and of nutrition; but, like other valuable discoveries, it now runs the risk of undue estimation, to be followed by a like depreciation. Thus, Prof. Bouchard[13] finds that three out of every ten hospital patients have gastroectasis, and ascribes to absorption from retained fermented matters the development of such diseases as phthisis, chlorosis, many nervous disorders, skin diseases, albuminuria, rickets and nodular rheumatism.

When due to organic stenosis of the pylorus, whether caused by thickening or cicatricial tissue from chronic ulceration or by cancerous growth, the dilatation is accompanied by hypertrophy of the stomach wall, as in other cases of hollow viscera like the bladder, large intestine, etc., where an obstacle exists to the evacuation of their contents. But simple dilatation of the stomach is characterized by a tendency to atrophy, involving more or less all the layers of gastric tissue, and is dependent upon (Allbutt[14]) the predominance of pressure upon the inner surface over the resistance of the walls of the stomach. This may occur from causes of constitutional weakness, as in patients with a phthisical or rheumatic diathesis. It often results also from the effects of continued fevers, notably typhoid, whose wasting effect, not only on the muscular wall, but also in producing mucous membrane atrophy of the stomach, is well shown by Fenwick.[14] In four cases that had died of this fever he carefully scraped off the whole of the mucous membrane of the stomach, and found that the average weight was only 580 grains, while the average weight of the gastric membrane in seventeen males who had died of other diseases was 1035 grains, showing that in this malady the stomach loses nearly half its bulk. There was, moreover, a like diminution of digestive power; for in seven cases of typhoid he made an artificial gastric juice of the mucous membrane of the stomach, and found that the average amount of albumin coagulated by it was only one grain, while the amount ordinarily dissolved by the same in those dying from other diseases was four grains. In two of the typhoid cases the albumin was quite unaffected by the artificial gastric juice.

When, therefore, convalescence from fevers seems to be too protracted and the patients complain of weight at the epigastrium, crustations of mucus or of wind, low spirits, loss of appetite and the tongue remains coated, but especially if the emaciation continues, examination for gastroectasis should not be neglected. Allbutt says that the disorder often occurs in laboring men as a result of local causes from their eating bulky, indigestible food washed down with great quantities of tea. This beverage is frequently a cause in servant-girls; and in the richer classes, dilatation follows the excessive use of ærated waters. Prof. Trastour,[15] of Nantes, maintains (with Bouchard) that women who are troubled with red noses, especially about the menopause, owe this unpleasant embellishment to fatty acids generated in dilated stomachs, with similar condition of the colon, which he maintains is frequently associated with gastroectasis from nervous paralysis, involving the colon as well as the stomach. To this gastro-colic dilatation he ascribes also the origin of many obstinate cutaneous disorders, besides many mental states of simple or emotional depression. Fr. Machon[16] ascribes most of these cases in children to congenital deficiency of the muscular wall of the stomach, and therefore particularly frequent in cases of rickets. They show at first great appetite and thirst, with persistent coated tongues and emaciation. Later they manifest a total repugnance to food of all kinds. Pain and a sense of weight at the epigastrium is constantly complained of. Vomiting is less frequent with them than with adults, but is fetid and greenish, very profuse in quantity, and shows contents which may have been retained for several days.

The clinical symptoms of this condition are slow digestion,—preceding for a long while all other developments. This in time gives rise to a sense of weight or of uneasiness at the epigastrium, with tenderness on pressure, and acid crustations, and finally to vomiting, which is characteristic. It often supervenes without antecedent nausea, is very profuse and comes with a gush. It is of a greenish or muddy appearance, with a fetid odor and filled with bubbles of gas. This vomiting may then not recur for two or more days, and then will show quantities of food which have been undigested for hours or davs. In these well-marked cases the face always looks worn, and emaciation sooner or later supervenes. Physical exploration is of the greatest value, but not without many difficulties. Inspection presents a soft, elastic tumefaction, whose outlines resemble that of the stomach. If the patient is emaciated, the peristaltic movements of the stomach (especially after a drink of cold water) may be detected through the abdominal walls; but Francon[17] maintains, and we think rightly, that this occurs only in cases where there is pyloric obstruction, and not in cases of simple dilatation. Percussion should next be resorted to, and the limits of this means of exploration are well defined by Pacanowsky,[18] who says that Penzoldt's method by pouring in water through the sound and then outlining the position of the dullness is accurate, but unpleasant and dangerous if ulcer be present. The same is true of Lente's process of feeling for the end of the introduced sound through the abdominal and gastric walls, and should never be employed when there are signs of ulcer, or when hæmatemesis has occurred in the history of the case. He also criticises Frerich's device of dilating the stomach with carbonic acid as not accurate or unattended with danger. This measure, however, is often quite conclusive of the presence of dilatation if it does not wholly succeed in defining its limits, and may be employed by directing the patient to take 6 grs. sod. bicarb., tartaric acid grs. 4, with two tumblers of water. Of the four boundaries to be determined, the lower is the most important for practical purposes. The others vary but little except when the stomach is distended by tartaric acid, and to determine them the patient should be lying on his back. The lower part of the right border cannot be determined by percussion, as it lies below the edge of the liver and to the right of the median line; the upper part of this border is easily determined by percussion. The left boundary cannot be located with certainty. Its upper border is partially covered by lung. Deep percussion shows the gastric limit, and light percussion that which the lung does not cover,—the first being usually at the lower border of the fifth rib. In dilatation it may reach the fourth or even the third rib. To determine the lower border the patient should be examined while recumbent to note whether the organ can be distinguished from the colon. Its percussion note is more tympanitic and less loud than that of the large intestine. The patient should then stand and drink half a litre of water, when a line of percussion dullness would be found, which would disappear when the patient again lay down. This line marked the lower border. In normal cases this line is above the navel 3–5 centimetres in men and 4–7 in women, and is never found in health below the navel. As succussion is employed in cases of suspected fluid in the thoracic cavity, so a splashing sound may often be elicited by fluid retained in a dilated stomach by swaying the body from the hips, but not by kneading the abdominal walls; for this precedure will frequently give rise to similar noises in chlorotics and others with much flatulent distension and functional paralysis of the intestines.

Treatment.—Prof. Ewald[9] recommends the administration of salol to determine the motor power of the stomach, or the period between the entrance of the food and its exit, based upon the fact that salol is not acted upon until it meets with the intestinal juice, when it soon after appears as salicylic acid in the urine. Salol was found by Ewald in twenty-five experiments to remain unchanged for hours when mixed with gastric contents, but dissolves rapidly in the alkaline secretions of the intestine. In healthy persons salicylic acid appears in the urine in one-half to one hour after the administration of salol; while in eight typical cases of dilatation of the stomach he found the appearance of salicylic acid delayed from two to three hours. In two obscure cases he was able to detect dilatation by means of this delayed reaction. This test is also useful in noting the effects of treatment; for by means of it Ewald has been enabled to determine that both electricity and massage hasten the passage of the chyme into the intestine.

In cases of long standing, and where the dilatation is so extensive that retention for hours allows of putrefactive or acid fermentation to occur, there is no measure which can compare with daily washing out of the stomach. The serious nature of all such retention is shown in chronic cystitis and in bronchiectasis; and it is hopeless to expect that the gastric mucous membrane, with its follicular structure of peptic glands, will not suffer even more severely from the corroding effects of putrid collections. Even vomiting, however copious, does not rid it sufficiently of the deleterious presence of such matters; for enough is always left to act as an immediate ferment for the next addition of food, not to mention the coating of viscid mucus which necessarily accumulates in all such inflammatory states of the lining of the hollow viscera. The unpleasant nature of this procedure is also much exaggerated; for I have myself got patients not only easily accustomed to it, but have had them learn to do it for themselves with only a little practice. All that is needed is a soft stomach catheter, which is to be moistened first with water, or preferably milk, and passed to the back of the throat over the left index finger placed on the tongue, the patient being told to swallow when the point of the catheter is passed down so that the constrictors of the pharynx grasp the end, and the instrument is then rapidly pushed on during repeated acts of swallowing. The tube must be passed at least twenty-eight inches, as eighteen inches represents the distance from the teeth to the cardiac end of the stomach. Penzoldt says that a tube one-third or three-eighths the length of the body suffices to reach the deepest part of the stomach. If there be much gas, the tube should be first introduced just within the cardiac orifice and the gas expelled through it by contraction of the abdominal walls, aided by kneading of the stomach. Lavage may now be begun by attaching to the catheter another piece of tubing three feet long by a piece of glass tubing, and a pint to a quart of water, tepid or cool, be poured in by a funnel at the level of the head, slowly and gently at first. It is then to be allowed to run out, and the operation repeated till it runs clear. If there be much mucus, a drachm and a half of sulphate of soda or bicarbonate of soda may be added to the quart; and if the contents are very fetid, 30–40 grains of salicylate of soda to the quart, or 1 per cent. of resorcin, may be used. The best time for the washing varies in different cases. If there be pain, and this is often the case at night, then the washing should be deferred till bedtime, the heaviest meal having been in the middle of the day, and but a slight meal taken in the evening. If, however, it be for better digestive action, it may be done some four hours after breakfast. The relief, by this procedure, of gastric pain is very striking, even in cases of fatal cancerous disease of the stomach. Dr. Benno Lewy[8] reports a case of gastric cancer, with atrophy of the mucous membrane and total absence of HCl, where the patient complained of torturing pains, which radiated to the spine and to the larynx, and which no remedies, including opium, relieved; but as soon as stomach lavage was employed they ceased at once, their recurrence being prevented for the rest of the patient's life by the immediate recourse to the washing whenever the pains began again. The editor has had two similar cases in this respect in his own practice.

A common result of gastric lavage is relief of the constipation, which is such a frequently accompanying symptom in cases of gastroectasis. It is probably due to suspension of intestinal peristalsis from the initial excitation of gastric movement being wanting. In Lewy's case, above cited, the constipation resisted the most drastic cathartics until the stomach washing was begun, after which the bowels never needed a cathartic. There are some precautions to be observed on the first employment of stomach washing, as the process cannot be said to be wholly without danger in a few cases. Thus, both Küssmaul and Lente speak of attacks of a more or less general convulsive character, following sometimes soon and sometimes a few hours after the operation, and particularly marked with general muscular rigidity. One case is reported[14] of a patient admitted into the Adenbrooke Hospital, under Dr. Bradbury for pyloric stricture. He was 48 years old, and had been an in-patient seven years previously, with symptoms of gastric ulcer. His stomach was now dilated and he suffered from flatulence, vomiting, pain and increasing weakness. On attempting stomach washing, soon after passing the tube into the stomach he became very faint, so that it was withdrawn. About two hours afterward he complained of stiffness of the jaws and rigidity of the arms, which were strongly pronated and flexed. The patient was conscious and sweated profusely. The rigidity spread to all the muscles of the limbs and trunk, and in six hours and a half after washing out of the stomach he died, the temperature having first risen to 107.2. Autopsy showed a pyloric stricture, with the scar of an old ulcer and a much-dilated stomach; but there was no injury or abrasion of the gastric mucous membrane. The other organs were healthy, and no lesion of the brain was discovered. In one of the editor's patients, an elderly man, who has derived much benefit from the lavage for the fourth time, and who had practiced it on himself without any apparent inconvenience, the patient was suddenly seized with a spasm of the throat in the night, some three hours after the washing, with no loss of consciousness, but with extension of rigidity to the arms. The attack subsided in half an hour, and he has had nothing similar since. Hence it is best to begin with gentle procedures and not elevate funnel above two feet until the stomach becomes more accustomed to the unusual sensations of the operation.

The medicinal treatment of gastroectasis is much the same as that of chronic gastritis. Lente, Penzoldt, and other German writers agree in recommending Carlsbad water, especially when taken hot, as the best for stimulating the movements of the stomach and intestinal walls, and for ridding the stomach of mucus. Matthieu[19] recommends strychnia, calabar bean, digitalis, and especially $\frac{3}{4}$ of a grain of ipecacuanha night and morning. The editor has found nothing so well borne as a mixture of carbonate of ammonia, grs. iv; ferri am. cit., grs. iij; tr. nux. vom., gtts. viij; tr. gentian, ʒj; elix. simpl., ʒj; aq. camphoræ, ad ℥j. The meals should be small and frequent, starchy and leguminous foods being avoided on account of their bulk, and fatty articles on account of the irritating character of the products of their fermentation.

DIET IN DYSPEPSIA.

The subject of diet in digestive troubles is of such interesting perennial concern that the journals of the past year abound in articles bearing upon it, but with not much that can be called new. Dr. J. W. Fraser[44] gives a résumé of a number of experiments made by him on the digestibility of the albuminoids, which were raw serum and egg-albumin, cooked albumin, globulins raw and cooked, myosin, and derived albumin, viz., syntonine, alkali albumin, caseine, and vegetable albumin or gluten. Each of these was subjected to six hours' peptic digestion, and then for six hours to pancreatic digestion. At the end of this time they were placed in a temperature of 80° C., and dialysis was allowed to proceed for twenty-four hours. These experiments show that raw serum and egg-albumin head the list in digestibility by pepsine. (1.97 and 1.96 per cent.) Next came syntonine or acid albumin (1.25 per cent.), which was made by submitting minced raw beef to hydrochloric acid solution, 4 drops of acid fort. to a pint of water, macerating over night, then draining off the liquid and boiling to remove the raw flavor. Myosin, raw and cooked, was quite high in the scale also (1.03 and 1.04 per cent.), while cheese (caseine), cooked albumin and vegetable albumin or gluten stood at the bottom. (0.47 to 0.60 per cent.) Cooked myosin ranked at the head in pancreatic digestion, while the raw albumin of serum and egg came next. The pancreatic juice acts especially well on raw albumin, and on acid and alkali albumin. Hence in fevers, when the gastric juice is secreted in very small amount, while the pancreatic gland continues active (Sir W. Roberts), raw albumin or syntonine or alkali albumin may be given alone or in beef tea. Ordinarily prepared beef tea is of little nutritive value; but if the white of an egg be mixed with a cup of beef tea and heated to about 160° F. the value of the beef tea is greatly enhanced. Dr. Fraser says "the experiments with caseine and gluten appear to show that there are very few worse foods for a delicate stomach than the usual bread and milk; for, as has been seen, the stomach only dissolves about 0.6 per cent. of these principles in the same time that it dissolves nearly 2 per cent. of serum albumin. And the caseine in the experiment was in an easier form for digestion than that in which it is usually presented to the stomach; for, as used, it was in a fine granular form, practically freed from fat with alcohol and ether; while cow's milk in the stomach forms dense, solid clots, rendered all the more insoluble by the fat, which forms the greater part of their bulk. True, the bread acts as a diluent to the clots, breaking them up, with passages for the gastric juice; but still it is evident that for digestion, by a delicate stomach, bread and milk does not form an ideal food. Whey contains lact albumin, which is the same as serum albumin, and it is a mildly nutritive food." Dujardin-Beaumetz,[45] of Paris, in the course of two lectures at the Cochin Hospital on the dietetic treatment of diseases of the stomach and intestine, with reference to gastric dyspepsia, says: "From the exclusive point of view of dietetics I divide dyspepsias into three groups,—dyspepsia by excess of gastric juice secretion, dyspepsia by deficiency of secretion, and dyspepsia with sympathetic troubles. For the first you will order a purely vegetable diet composed of bread, vegetables and fruits. For drink you will order milk, and you will sometimes allow beer, but never wine. For dyspepsia with deficiency of gastric secretion, meat and soups are the best peptogenous agents. The meat should be in the most assimilable form, in the form of powder or pulp; but be careful that the amount given is proportioned to the digestive power of the patient. Milk also is beneficial, especially fermented milk, on account of the lactic acid which it contains. As wine and alcohol augment the acidity of the gastric juice, you may give wine or brandy and water. For dyspeptics with sympathetic disturbances, such as gastric vertigo, you should diminish all the excitations which result from the irritation of the mucous membrane of the stomach; and you will be most likely to do so by making your patient a vegetarian, by restricting him to a regimen of bread, fruits, legumes and vegetables, with milk for a drink. Order the crust of bread rather than the crumb; or, better still, bread well toasted. All vegetables must be well boiled, while fruits must be baked or stewed, grapes alone being excepted. In this dietary I allow eggs, on condition that they be soft-boiled or taken raw. For drinks, beer seems to me preferable to wine, in a strictly vegetable regimen. In the dietetics of dilatation of the stomach it must be insisted upon that the patient should not drink between meals; and, with Bouchard, I would limit the amount of fluid to only 12 fluidounces a day, with no wine. All fatty substances must be avoided, only bread crust or toast; and there should be nine hours between breakfast and dinner, and fifteen between dinner and breakfast. For those who with gastroectasis have diarrhœa, I absolutely cut off meat and eggs, and limit them to starchy foods, vegetables and fruits. They may take Bavarian or Pilsen beer, or liquid malt. I add to this purely vegetable regimen, the employment of carbon bisulphide water, which has done me great service:—

℞	Pure sulphide of carbon, ...	f ʒvj
	Ess. menth. pip., ...	gtt. j
	Aq., ...	℥xiij

"Mix in a flask, shake, and let settle. Use the clear supernatant solution, and renew the water as fast as you pour off from the flask for medicinal use. I give, daily, from four to eight tablespoons of this solution at meal times and between meals. Each dose is taken with half a glass of milk or beer. In case of patients with gastroectasis who are constipated, I permit roast meats, and order toasted bread and fruits,—especially peaches and grapes."

As to beverages, Dr. H. A. Hare[6] communicates a list of tests of the influence on peptic digestion of some twenty different brands of American and German lager beer. Considerable differences were noted, amounting to as much as fifty-nine minutes in the length of time between the different varieties of these beers of their delaying peptic digestion in the test tubes; but as, in all cases, the delay is to be ascribed to the alcohol present, and no account is taken of the bitter principles which they contain, which may in the stomach stimulate digestion, Dr. Hare's conclusion is that most such beers do not retard stomach digestion at all; while others, whose interference with peptic action is very slight, may greatly promote it. Thus, Bergner & Engel's Philadelphia Tannhäuser beer delayed peptic solution only one minute and thirty-seconds; while Clausen's New York champagne lager, Budweiss, Kaiser, of Bremen, and Best, of Milwaukee, were each under ten minutes in their effect. English beers being, of course, much heavier, contain more alcohol, but also many nutritive matters,—which cannot be said of lager beer, since its chief nourishing constituent is glucose, which is, however, in very small amount.

Dr. T. W. Fraser[46] contributes an article on the influence of tea, coffee and cocoa on digestion, in which he made use of the same tests for peptic and pancreatic digestion, and then of dialysis, as in the case of foods above mentioned. The results show that all the three beverages retard the digestion and absorption of all the nitrogenized proximate principles of dietetic substances, although tea may assist the diffusion of peptones from the stomach. Neither of them, therefore, compare with water as a standard for experimental investigations. He concludes therefore: "(1) That it is better not to eat most albuminoid food-stuffs at the same time as infused beverages are taken; for it has been shown that their digestion will in most cases be retarded, though there are possibly exceptions. Absorption may be rendered more rapid, but there is a loss of nutritive substance. On the other hand, the digestion of starchy food appears to be assisted by tea and coffee and gluten. The albuminoid of flour has been seen to be the principle least retarded in digestion by tea, and it only comes third with cocoa; while coffee has apparently a much greater retarding action on it. From this it appears that bread is the natural accompaniment of tea and cocoa when used as the beverages at a meal. Perhaps the action of coffee is the reason why, in this country, it is usually drunk alone or at breakfast,—a meal which consists much of meat, and of meats (eggs and salt meats) which are not much retarded in digestion by coffee. (2) That eggs are the best form of animal food to be taken along with infused beverages, and that apparently they are best lightly boiled if tea, hard boiled if coffee or cocoa, is the beverage. (3) That the caseine of the milk and cream, taken with the beverages, is probably absorbed in a large degree from the stomach. (4) That the butter used with the bread undergoes digestion more slowly in presence of tea, but more quickly in presence of coffee or cocoa; that is, if the fats of butter are influenced in a similar way to oleine. (5) The use of coffee or cocoa as excipients for cod-liver oil, etc., appears not only to depend on their pronounced tastes, but also on their action in assisting the digestion of fats."

GASTRALGIA.

Sir James Sawyer,[14] in a lecture on this subject, defines gastralgia as an affection in which pain, deep-seated and paroxysmal, in or about the stomach, of a neuralgic character, is the leading symptom. The pain—generally very violent, with a sense of contraction at the pit of the stomach—comes on suddenly or after being preceded by a sense of oppression. It generally extends to the back, with a sense of fainting, the face falling in, the hands and feet growing cold, and the pulse small and intermittent. It is common to find pulsation at the epigastrium, and pressure is not only well borne, but the patient often forces the pit of the stomach against some firm object, or compresses it with his hands. In many instances sympathetic sensations occur in the thorax, under the sternum, or in the pharyngeal branches of the sympathetic, while they are seldom met with in the superficial parts. As to other aids to diagnosis Sawyer points out that (1) gastralgia is not a wasting disease; (2) it is not safe to diagnose cancer of the stomach till you can feel the cancer; (3) nor should ulcer of the stomach be diagnosed till blood from the stomach has been seen, either in hæmatemesis or melæna. It may be assumed that pain arising in the stomach when the organ is empty, and relieved by the ingestion of food, is of nervous origin. While gastralgia may occur at any age, it is commonest in middle life, especially in women with anæmia, hæmorrhages or prolonged uterine discharges. In the treatment, Sawyer is pronounced in favor of arsenic as a specific for this affection. He gives $\frac{1}{24}$ of a grain of arsenious acid in pill with two grains of extract of gentian thrice daily between meals, and continues for a few weeks. In severer cases he adds counter-irritation to the epigastrium by a fly-blister kept open by savine ointment. The diet should be generous. A dyspeptic regimen makes a case of gastralgia worse.

GASTRIC ULCER.

The much-disputed question of the etiology of Cruveilhier's round ulcer of the stomach is the subject of an able paper by Marfan,[20] who describes first the erosive and ulcerative affections of the stomach other than the round ulcer, to exclude their relation to it either as regards origin or process of development. (1) Erosions are common in the course of different chronic gastric inflammations, and occur under the two forms of follicular and of hæmorrhagic erosions. The follicular variety (Brinton, Wilson Fox) occurs in scattered points about the size of a pin's head, and most numerously about the pyloric region. Fox explains them as the result of infiltration and ulceration of glands similar to the solitary glands in the intestine, and they are to be found chiefly in patients in the latter stages of phthisis. Hæmorrhagic erosions are described by Brinton as arising from congestive states of the mucous membrane, and as occurring in spots about the size of a mustard-seed to that of a pea. Balzer, however, maintains that they always begin as minute abscesses, which are enlarged after rupture by the action of the gastric juice. Their symptoms are sensitiveness to epigastric pressure, xiphoito-vertebral pain increased after food, and finally vomiting intermixed with streaks of blood. It is in alcoholic gastritis especially that they occur, and they are cured by the cessation of drinking habits. Treitz and Luton describe them as occurring in uræmic gastritis, and Chauffard in typhoid fever.

True ulcerative conditions of the stomach, when produced by known processes other than the round ulcer, are rare; and hence by contrast of their characters serve to exclude certain supposable causes of the origin of the latter. (1) Ulcers caused by corrosive poisons, such as the mineral acids, arsenic, ammonia, etc., produce gastric lesions very variable in extent and situation, and so ill-defined in outline that they cannot be specifically described as the round ulcer can. (2) Alcoholism produces ulcerative changes in the stomach which are always consecutive to fatty degeneration of the glandular tissue, and begin as whitish patches not unlike in appearance those of muguet. These in turn seem to be attacked by the gastric juice; but the resulting ulcers differ from the round by their puckered appearance, their slight depth, and their number. They are always accompanied by pain, often paroxysmal, especially after eating, and frequently by hæmatemesis; but are quite curable on suspending the use of alcohol. (3) Typhoid fever gastric ulcers are sometimes both extensive and deep, with irregular borders. They may be due to venous thrombosis; but their distinguishing feature is their dependence on this fever. (4) Tuberculous ulcers of the stomach are quite rare, and are so constantly associated with similar ulceration of the intestine as to lend color to the theory that they result from an ascending process from the intestine. (5) Klebs and Cornil have described syphilitic gummata of the stomach, generally small and round, but occupying the lesser curvature and near the cardia. They, however, are rare. (6) Ulcers in the stomach occurring during acute pemphigus, in lymphadenoma, in variola, and as a kind of phlegmonous gastritis, have been described by various authors; but in each their characters were quite unlike those of the common round ulcer.

In marked contrast to all these we have the great preponderance as to frequency of the single round ulcer; and its chief etiological factors are equally distinct from any of the causes of ulceration above enumerated. (1) The influence of age, its maximum period falling between twenty and thirty years of age. (2) Sex. Dr. Brinton gives the proportion of two women to one man, and Willigt of seven women to two men. (3) The chlorotic condition among the women, for much the greater number of them are fully developed cases of chlorosis, or have long suffered from irregular menstruation or deficient appetite and imperfect digestion. Those writers who, like Brinton, regard the round ulcer as starting from some initial superficial erosion or ulceration which is afterward deepened and rounded in its contour by the digestive action of the gastric juice, have to explain why, when many erosions or hæmorrhagic ulcerations occur, as above described, in alcoholic gastritis, etc., none, on the one hand, develop into a round ulcer by this means; nor, on the other hand, why, when a round ulcer is found, it is single, without any small hæmorrhagic ulcerations existing beside it. The observations of Potain and Duplay, of ulcerations resembling the round ulcer occurring after a variable interval after contusions of the stomach, do not throw much light upon the subject; because in every case of the kind the ulcers soon healed completely,—which is quite unlike the persistence and progressive march of Cruveilhier's malady. The same may be said of Quincke's experiments on dogs with gastric fistulas, in which he produced round ulcers by a great variety of injuries of the gastric mucous membrane; but he adds that they all healed again with remarkable rapidity.

Virchow's well-known theory of local embolism or thrombosis, causing an initial necrosis of a mucous membrane area by arrest of blood supply, and that the gastric juice does the rest in forming the round ulcer, has had many advocates. The experiments of de Prévost and Cotard, in which they caused intestinal ulcers by artificial embolisms induced by injecting tobacco-seeds in the aorta, seem to support this hypothesis, except that they never succeeded in producing a single gastric lesion by this means. Meantime there are serious anatomical difficulties toward accepting this theory; for the anastomoses of the terminal gastric arteries are so exceptionally abundant that it seems most improbable that the blood supply of any gastric area can be suddenly obliterated, either by a single large embolus or by many small emboli. Cruveilhier's own view, that the round ulcer is the result of a chronic gastritis only, is opposed by the fact that severe and chronic gastritis is a very common condition in both men and women of all ages, and especially after middle life, without any round ulcers developing in its course; and hence this theory does not account for its frequency in young adults or in chlorotics. It is evident, therefore, that there must be some special, and perhaps specific, predisposing cause for this intractable affection, of whose precise character we are still ignorant. Riegel regards a constant state of hyperacidity as the chief cause.

Dr. Decker,[8] of Wurzburg, raises the question whether taking hot drinks may not initiate the lesion which develops into the round ulcer. He adduces two experiments of his on dogs to prove this view, and cites the clinical fact of the frequency of this disease in cooks, who habitually test the flavor of their dishes when very hot. It has often occurred to the editor, especially in hospital practice, to note its frequency in domestics who are given to tea drinking, often on empty stomachs. This habit, moreover, is peculiarly apt to cause acid dyspepsia, heart-burn and palpitation, and in so far lends color to Riegel's theory of the part that hyperacidity has to play in the development of the affection. For the treatment of these cases the editor has not found any thing better than bismuth in powder, with small doses of opium, to be continuously followed by nitrate of silver in solution ¼ gr., with a small dose of laudanum, and rest to the stomach.

GASTRIC CANCER.

No question in pathology has been more widely discussed on the Continent of Europe during the past year than the absence of free HCl from the stomach in cancer of that organ, to which attention was first drawn by Van der Welden.[18] In Germany, Riegel, Ewald, Kraus, Boas, Cahn and V. Mering and many others have contributed papers on the subject, along with Debove, Renault, Lépine, Dujardin-Beaumetz, in France; Sansoni in Italy, etc.; and the general result, while modifying Van der Welden's dictum that such absence is proof of the existence of cancer in the stomach, yet undoubtedly leaves it as one of the most constant symptoms of that affection, and hence of high, though not of pathognomonic, significance.

In this investigation the question of chemical tests is of course of primary importance. The presence of free acid in the stomach is shown by adding to the suspected fluid a saturated solution of tropælin. This turns to a deep brownish-red with free acids, and to a light yellow with acid salts. For free lactic acid the test is to add a mixture of carbolic acid to ferric chloride diluted with water until an amethyst blue liquid is obtained, when, on the addition of a fluid containing lactic acid, it turns to a greenish yellow. For the detection of hydrochloric acid the directions of Riegel are as follow: (1) The stomach contents are not to be examined until three hours after eating. (2) Vomited matters are not suitable, because the chemical reactions are disturbed by large quantities of bile and mucus. The gastric contents are withdrawn by the siphon, and then the gastric secretion is first stimulated by the introduction of 100 c.c. of cold water, to be followed in ten minutes by 3 c.c. of ice water. Then the whole is collected, and, after first determining the presence or absence of HCl, the digestive power on albumin of the liquid is ascertained. The test for HCl is thus employed: Into each of two test tubes are poured 50 c.c. of distilled water, followed by two drops of a 2 per cent. solution of methyl violet. Into one of the tubes is now added, drop by drop, the fluid obtained from the stomach, and to the other an equal quantity of pure water. If free HCl be present the first tube will turn blue, while the second will retain its violet hue. Riegel declares that in several thousand specimens of gastric juice he did not encounter one that digested well where this reaction failed; and, on the other hand, every specimen which gave this reaction also possessed digestive power.

Cahn and von Mering,[18] however, pronounce this methyl test untrustworthy, because not only neutral chloride solutions, the acetate of soda, and the magnesia mixture for testing for phosphoric acid may produce a blue with this test, but also certain neutralized fluids obtained from the stomach, also dissolved peptones and amide acids, and to a less degree mucin. Instead of this they propose a complicated test for HCl by litarmetric methods: first, getting rid of the volatile acids by distillation and of lactic acid by ether, and then testing for HCl by cinchonine shaken up in chloroform, which separates the chlorides; then distilling this chloroform extract, dissolving the residue in water acidulated with acetic acid; then adding nitric acid, and testing finally for chlorine by silver nitrate. With this test they claim that in five cases of pyloric cancer, where the diagnosis was confirmed by the autopsy, they found hydrochloric acid in every instance, and that its presence was the rule and its absence the exception. C. Honigmann and C. v. Noorden[22] confirm the accuracy of Cahn and von Mering's litrate method, but deny that the acid found was HCl, because it possessed no peptic strength, that their acid reaction was due instead to acid compounds, the small amount of HCl secreted in gastric cancer having united with albumin compounds and been absorbed. Free HCl is not, therefore, found in these cases, and hence the methyl blue reaction is trustworthy, because it will not occur unless free HCl is present. According to Renault, gentian violet will give as good a reaction in testing for free HCl as methyl violet. Recently, Dr. Alfred Günzberg, of Frankfort-on-the-Main, who regards gentian violet as the best of the substances hitherto used for the detection of HCl, objects to it because it gives the blue reaction if organic acids, especially lactic acid, be present, and he substitutes for it 30 grains of phloroglucin and 15 grains of Merck's vanilline in about an ounce of absolute alcohol making a yellowish-red solution. A trace of concentrated mineral acid added to this solution produces a bright red color, and at the same time there takes place a separation of beautiful red crystals. Concentrated organic acids (lactic or acetic), either alone or mingled with chlorides, give no such reaction. If diluted acid be used, the color does not appear until some of the fluid is driven away by heat, when the red crystals appear. Boiling must be carefully avoided. This test is then used thus: A few drops of the filtrate of the stomach secretion are mingled in a porcelain dish with the same quantity of the phloroglucin-vanilline solution and carefully heated. The reaction will then invariably take place if $\frac{1}{10}$ of 1 per cent. of free HCl be present,—a test, at least, equal to that of the aniline solutions. If but $\frac{1}{20}$ of 1 per cent. be present, the reaction occurs in the form of red streaks, and below this degree it ceases altogether. Hence he thinks that an approximation to a quantitative estimate of the HCl present may be made in accordance with the intensity of the reaction.

The significance of the absence of free HCl depends, however, on conditions of the stomach and of the general system which occur almost invariably in cancerous disease of the stomach, but also occasionally in other affections. Riegel[22] reports his examinations in 16 cases of gastric cancer, in 5 of which the diagnosis was confirmed by autopsy. In these 5 he made 154 analyses, and never found free HCl in them, nor did the stomach filtrate possess any digestive power. In 1 he gave large quantities of HCl soon after meals; but on testing no free HCl was found, and the gastric juice was without digestive power. In 8 of the remaining 11 cases, 120 analyses failed to find either free HCl or digestive power. In the remaining 3 there was found at first a very weak HCl reaction and feeble digestive power, and they were, therefore, regarded as beginning cases of carcinoma; for later on no free HCl was found. From these observations Riegel concludes that the gastric juice of a cancerous stomach has neither free HCl nor digestive power; and also that if any given case of gastric disease should show a stomach secretion with free HCl in it, and that it possessed digestive power, he would exclude carcinoma in spite of the most decided symptoms of that malady. Riegel, however, claims that he was the first to show that HCl may be absent in other affections besides cancer, such as amyloid disease of the mucosa, toxic gastritis and continued reflux of bile. Meantime Prof. Ewald with Dr. L. Wolff[8] report 8 cases, some of them in good health and others with only slight gastric disturbance, in whom there was absence of HCl even after administering ʒss doses of ac. hydrochlor. dil., from which they conclude that this acid may be absent from the stomachs of perfectly healthy persons. Grundizach, in the same journal, reports having found five among several hundred patients without cancer in whom HCl was absent even during digestion, but in whom there was a great deal of mucus secreted, owing to gastric catarrh. Dr. Boas[23] finds that absence of HCl may occur (1) in fever; (2) carcinoma of the stomach with dilatation; (3) chronic gastric catarrh; (4) atrophy of the gastric mucous membrane, as in pernicious anæmia; (5) intense general marasmus. While Riegel considers the absence of HCl in cancer as dependent upon the cancerous cachexia itself, Ewald upon the decrease of the energy of the stomach from general constitutional enfeeblement, Boas ascribes it chiefly to the chronic gastric catarrh which invariably accompanies cancer of the stomach. With reference to gastric catarrh, he recognizes two stages, in the first of which, where the disease has not made much progress, free HCl is found; but in the second stage, when increased changes of texture have occurred, such as atrophy of the glands and proliferation of interstitial tissue, the HCl may be reduced until not a trace of it is found. Dr. A. Kirstein,[24] assistant at Prof. Rossbach's clinic at Jena, reports the case of a man who appeared to be suffering from gastric cancer. The contents of the stomach were examined repeatedly, on different days and at different hours, and the methyl violet reaction to HCl was plainly obtained. Yet after death the autopsy showed the presence of a gelatinous carcinoma of the mucous membane, with metastatic nodules in the peritoneum.

Renault[26] says that Jaccoud always examined the glands in the groins in a case of suspected cancer of the stomach, while Henoch drew attention to the diagnostic importance of enlargement of the supra-clavicular glands in the same affection. These glands, however, have been found enlarged in cancer of the œsophagus, of the pleura, of the lungs, and even of the uterus; but, in the absence of disease in those parts, this sign may be of diagnostic value when there are gastric symptoms present. Quinquaud points out that in the differential diagnosis between gastric dilatation and cancer the hæmaglobin of the blood is reduced to a much lower point in the latter, while the blood corpuscles are more irregular and crenated, as in pernicious anæmia. The great diminution of urea excretion noted by Romnelvere, of Brussels, in cancerous diseases, is of importance, though found in other diseases. The editor had one case in which the proportion was much below 150 grains per diem, with unaffected kidneys.

Treatment.—Recently Dr. L. Riess[8] has published a long list of clinical histories of the beneficial effects of condurango bark in this disease, in which he declares that the painful symptoms of gastric cancer in cases where the autopsy proved the presence of the malady, were removed by its use; and that other affections of the stomach with a train of symptoms proper to cancer have been sensibly relieved, and in some even cured. By the employment daily of 150 grains of condurango continued for some months, the appetite is seen very shortly to be improved, nausea and vomiting diminished, pain removed and the strength and weight increased. In all cases the duration of life has been lengthened; but the most remarkable result has been in the influence of the remedy upon tumors in the stomach which have been accessible to palpation. In a total of 64 cases a sensible diminution of the tumor could be demonstrated 17 times; in 8 other cases the tumors disappeared completely or nearly so, and in the remainder the tumors ceased to increase in the same proportion as formerly. Orszewsky and Erichson[27] have reported such a case in which the tumor disappeared under the use of condurango, and which after death seemed to have been reduced to a mere thickening. Their theory is that condurango has the property of favoring the development of connective tissue in the tumor in the walls of the stomach, and at the same time destroying its cellular elements. That it must produce some local influence on the tumor itself Reiss infers, partly from the fact that it has very little influence on remoter cancers, and partly because the cases the most benefited by it possessed tumors in the pylorus or very close to it, enabling the remedy to be longer in contact with the disease, especially if complicated with stenosis of the pylorus and dilatation of the stomach, as it so often is. Riess gave much larger doses and oftener repeated than Friedreich. His mode of administration was daily a decoction of 150 grains, with five drachms of syr. simp., aq. ad ℥vj., a tablespoonful every hour or two hours. As examples of the long continued administration of the drug, he mentions two women, one of whom took 800, the other 900 grams; and three men who took 830, 870, and the third about 1000 grams of the condurango without interruption.

DISEASES OF THE PANCREAS.

ORGANIC DISEASE.

Prof. E. Küster[23] read a paper before the Berlin Medical Society on cysts of the pancreas, in connection with a case of his own on which he had successfully operated. He could find only 11 cases of such operations reported, in 9 of which a wrong diagnosis had been made. When pancreatic cysts form appreciable tumors the mistakes of diagnosis may vary according to sex. With men they may be taken for hydatid cysts of the liver, mesentery or kidney, lymphatic cysts, or aneurisms of the aorta or of its branches; with women they may be easily confounded with ovarian or uterine cysts. Of the 11 which he had collated, 6 were women; and in 5 of them the tumors had been supposed to be ovarian, while one was taken for a hydatid cyst of the liver. Pancreatic cysts should be distinguished from ovarian by their development from above downward, while ovarian develop from below upward. If the cyst has not contracted extensive adhesions with the liver, it will not be displaced by the respiratory movements. Renal cysts but rarely attain the dimensions which would cause them to appear in the locality where pancreatic cysts develop, while aneurisms are to be made out by their well-known signs. Exploratory aspiration of the contained fluid is of considerable importance. The fluid often contains blood, like cysts elsewhere, but in addition is quite albuminous, and if a sufficient quantity is obtained will show strong digestive power. The absence of the hooklets, etc., will serve to distinguish it from hydatid fluid, and its characters already mentioned from false lymphatic cysts, whose contents are usually little else than feebly saline water. Of the clinical symptoms of pancreatic tumors the most striking is pain, owing to the early implication of the cœliac plexus. Along with this the stomach is often much disturbed, and there is frequent vomiting. Rapid emaciation is also reported in some, probably owing to the irritation of the abdominal sympathetic, and several authors have mentioned diabetes as a complication. In his case there were signs of diabetes insipidus. The presence of undigested fats in the fæces he found to a slight degree in his case; but he lays little stress upon this supposed symptom of pancreatic disease, as the presence of undigested fat in the pus is mentioned only exceptionally in recorded cases of affections of the pancreas, often from malignant disease or from cysts. He found, however, in his case much plainer evidence in the fæces of undigested nitrogenous than of fatty matters. The want of fat digestion might, however, have been overlooked in most of the cases collated, because disease of the pancreas was so little suspected; and as, moreover, some of the secreting structure of the gland is generally still functionally active in cystic disease, a good part of the fats ingested may have been acted upon and absorbed.

Treatment.—As to treatment, we may resort (1) to simple aspiration or puncture; (2) total extirpation; or (3) fixation by suture of the sac to the abdominal parietes. Puncture is not cure, and, moreover, is not without its dangers. Extirpation is scarcely possible, because the cysts are generally extensively adherent to the surrounding organs. Five such operations were attempted, and only one recovered. Six times the sac was incised and then attached by suture to the abdominal walls; five of them recovered and one remained with a permanent fistula. Such a fistula is very troublesome, and is due to some portion of the gland being left in the cystic wall, so that it continues to secrete. On this account remains of the gland in the sac should be carefully sought for during the operation and wholly removed.

Dr. E. L. Call[28] gives the history of an interesting case of chronic pancreatis in a lady æt. 62, widow, first seen Feb., 1886, and who died a year afterward, having progressively declined with symptoms of great debility, breathlessness on the slightest exertion, complete anorexia, very sallow skin, œdema of the feet, and occasional vomiting, especially after any unusual movement. For a time pernicious anæmia was suspected, but the red corpuscles were not altered in form. In August she had a sharp attack of hepatic colic, followed by intense jaundice, from which she recovered but slowly. After a similar attack in October, when she passed a gall-stone the size of a pea, the digestive disturbances were more marked. She complained constantly of a burning pain under the lower end of the sternum coming on two hours after eating. She also complained of a feeling of weight in the bowels when she sat up, as if some heavy body was dragging from the spine. Her bowels were very irregular in action and in the character of the stools; but though every stool was examined by her daughter, an unusually intelligent person, and frequently by Dr. Call himself, yet no fatty matters were ever found in them. She never had another paroxysm of colic, but the digestive distress increased. She became extraordinarily emaciated, and during the last month of her life she was constantly disturbed by twitching of the feet and legs, and sometimes of the arms; and for two weeks before death she had to be kept constantly under sedatives.

The autopsy showed the stomach walls to be very thin, but there was no trace of malignant disease, either in that organ or in the duodenum; intestines very anæmic, small intestines contracted, large intestine somewhat distended, and the walls in places so thin as to be almost transparent; no peritoneal adhesions or signs of inflammation; liver slightly enlarged, pale, yellowish, and of firm texture; gall-bladder the size of a large pear, containing three large and many small gall-stones. Upon raising the stomach the pancreas was seen larger than normal, dark gray in color, and so firmly adherent to the vertebræ and posterior abdominal walls that it was detached with great difficulty. It then looked as if there must be malignant disease of the organ; but on immersion in alcohol it was found impossible to harden it, and it shrank greatly in bulk, while large quantities of fatty matters were dissolved out of it into the alcohol. On microscopical examination there were found a large increase of connective tissue, an abundant deposit of pigment, considerable fatty degeneration, and very little trace of acini or glandular epithelium.

This case is interesting because in the majority of reported cases of pancreatic disease there was carcinoma, rendering it difficult to separate the symptoms, which might be due either to cancer in general or to the implication of neighboring organs in the course of the disease. The symptom of a dragging sensation from the spine, especially on assuming the erect position, is mentioned by several authors as a sign of organic pancreatic disease. The nervous distress and twitchings may be explicable from the manner in which the solar plexus was involved, and this may account also for the vomiting and the digestive disturbances. The atrophy of the intestinal walls may have been occasioned by the superior mesenteric artery being much compressed near its origin by the vertebral adhesions; for in this case the artery was given off above the pancreas, and ran across its whole posterior surface so as to be involved in the fibrous bands which attached the gland to the vertebræ. At no time during life was sugar found in the urine.

It is evident from such cases that it is always more or less difficult to interpret the symptoms of organic affections of the pancreas, or to derive from them any certain conclusions as to the functions of the gland in health. It is familiar to all who have had extensive clinical experience that emaciation is quite as pronounced after simple obstruction to the flow of bile as in cases of pancreatic disorder; while on the other hand the implication of the solar plexus may explain much of the digestive disturbance and the malnutrition, irrespective of the arrest of the pancreatic secretion. In the discussion on the causation of the bronzing of the skin in Addison's disease, some writers have advocated the view that the discoloration is not from perverted function of the adrenals, but because tubercular disease of those glands frequently causes inflammatory adhesions to involve the neighboring solar plexus. Dr. Dyson[29] reported a case to the Sheffield Medico-Chirurgical Society of a hospital patient who, when he was admitted, had a discoloration very similar to Addison's disease, and who after death proved to have cancer of the head of the pancreas with extensive implication of the liver and the surrounding parts.

FUNCTIONAL DISEASE.

In an important memoir on the presence of fats in the digestion of icteric patients, and the changes in those fats, if there be no interference with the flow of the pancreatic juice, Müller shows that while the absence of bile in the intestine does not interfere with the absorption of amylaceous matters, and diminishes but slightly that of the albuminoids, it interferes to a great extent with the absorption of fats. He then shows that the whitish stools of jaundice owe their color not only to the absence of bile pigment, but also to the large amount of unabsorbed fats. In healthy persons from 6–10 per cent. of the fats ingested are to be found in the fæces; in cases of biliary obstruction the proportion rises to 52–78 per cent. With healthy adults the fats in the fæces occur in the form of irregularly polygonal vitreous plates, formed in small part of neutral fats, but mainly of soaps,—the yellow calcareous crystals of Nothnagel. Drops of free fats are not found in healthy fæces unless oils of low melting point have been freely administered, as cod-liver or castor oil, or else when the absorptive power of the intestine is much diminished.

With icteric patients, however, peculiarly formed needle-like crystals of fat are found, as first observed by Nothnagel and by Gerhardt. The first of these are free fatty acids, in long, feathery, tufted crystals, easily dissolved in ether and resolved into fine drops by heat. The second forms are saponified fats, much shorter, some rectangular and terminating in less pointed angles, and massed in bundles or aigrettes. They resist the action of both ether and heat; attacked by acids, these soaps give up numerous droplets which are soluble in ether. Now, when these fatty crystalline bodies are found largely in the fæces, it is a sign of some interference with the absorption of fats in the intestine. By a singular chance, Müller had the opportunity of an autopsy on a patient with jaundice whom he had watched during life, and in whom the pancreatic duct opened independently into the intestine about one centimetre from the orifice of the bile duct. The pancreatic duct was permeable and the pancreas itself healthy. In this case the fats in the fæces were 47.8 per cent., of which 31.4 per cent. were neutral and acid fats, and 68.6 per cent. soaps; and on microscopic examination there were great numbers of soap crystals, which showed that the greater part of the unabsorbed fats had been altered, as he concludes, by the pancreatic juice, but not made absorbable owing to the deficiency of bile. He would assign to the pancreatic juice, therefore, the rôle of acting on the fats, but not enough without the bile to render them absorbable. Strümpell[30] relates an experiment of his confirmatory of the color of icteric fæces being due to the presence of fats, in which he gave to a patient with intense icterus food made as free as possible of all fats, and observed that the dejecta were thin, of a clear tint, but no longer clay colored.

O. Gerhard[31] reports a very interesting case as regards this question, of a patient admitted to Prof. Eichhorst's clinic, at Zurich, with pain about the navel, vomiting of small quantities of bilious and mucous fluids, slight icterus, continued closure of the bowels, no discharge of flatus, but slight distension of the abdomen and no increase of indican in the urine. Laparotomy was performed on the sixth day of his illness, without finding the seat of the obstruction. Patient died on the ninth day of his illness, and the autopsy showed compression of the duodenum by a greatly enlarged, partially necrosed and hæmorrhagic pancreas. The diagnosis was intestinal obstruction, although most probably of the large intestine. Absence of fæcal vomiting and of extensive meteorism rendered this conclusion uncertain. The autopsy, however, cleared the question; for the high location of the stenosis explained why there was no fæcal vomiting, while the absence of the pancreatic juice, from disease of the gland itself, prevented the formation of the indol on which the above test depends.

DISEASES OF THE LIVER.

CATARRHAL JAUNDICE.

Heitler[35] contributes several elaborate articles on cases of catarrhal jaundice which occur as accompaniments of some general morbid condition, and hence are to be regarded as indications not of a local hepatic disorder only, but of some constitutional disease. In many of these cases the jaundice itself may be only slight or temporary, in others progressively severe, but in all evidences of the implication of other organs will soon become pronounced. He would divide the cases of catarrhal jaundice, therefore, into the two classes of local and constitutional icterus. The latter are to be distinguished by concomitant enlargement of the spleen. In opposition to Maragliano,[18] who ascribes the splenic engorgement to a mechanical reflex into the splenic veins from portal obstruction, he, as well as Chauffard, shows that this does not occur in cirrhosis when the portal obstruction is at its highest; nor in acute local catarrhal jaundice, impaction, etc. Other concomitants are pneumonia and nephritis, each frequently with a quasi-epidemic character in conjunction with the icterus. Hence the prognosis of catarrhal jaundice must often materially differ, according to whether it be or be not accompanied by signs of simultaneous rather than consecutive affections of other organs.

Treatment.—A method of treating catarrhal jaundice introduced several years ago by Krull, by free cold water injections, is rapidly gaining favor. Reports by Lowenthal, Eichhorst, Krauss and others speak very highly of its cutting short the complaint sooner than any other method. Chauffard[1] speaks very enthusiastically about it, and records seven cases treated by this method alone. He found that one injection a day was enough after the procedure of Krull; for in those in which he tried them twice a day the recovery was not any sooner. The patient should lie down on the left side, and the first day water at 59° F. should be injected, from one to two quarts; on the following days the temperature may be gradually raised to 72°. The injections cause some colicky pains from the active peristaltic movements set up, but are well borne and retained from five to ten minutes, and after the passage the patient usually feels considerably relieved. Improvement begins almost at once. Lowenthal claims that only four injections are needed; but in proportion to the antecedent length of the case they may have to be continued longer. After beginning this treatment the pruritus and yellow vision disappear with great rapidity, the fæces resume their natural color, and the bile pigments disappear from the urine in from two to eight days. Musser,[5] of Philadelphia, also speaks well of this procedure. The editor has tried it in cases at Bellevue and Roosevelt Hospitals with but moderate success, probably because the water used was not cold enough. Chauffard warns against its use in obstructive jaundice, as in one case of the kind it caused much distress, though not in the form of severe colic.

GALL-STONES.

Gall-stone Cholesterine not from the Bile.—Dr. J. Syer Bristowe[14] reported a case of a female patient aged thirty-eight, who had been jaundiced for twenty years, the jaundice varying in intensity, but never quite disappearing. As long as she could remember, she had been liable to "spasms" commencing in the left hypochondriac region and extending thence obliquely into the right lumbar region. She finally had an attack of "spasms," lasting for ten days, which left her very weak, after which she first discovered a small lump on the right side, which grew into a constantly enlarging, painful tumor, whereupon she was admitted to the hospital. Her urine contained neither sugar, albumin nor bile. The tumor encroached on the skin, and an exploratory incision being made, showed a few drops of pus in an indurated, infiltrated tissue, whence after a few days, several black, rounded bodies issued in the poultice applied to the part. They were insoluble in ether and chloroform, and with nitric acid gave the reaction of bile. An incision into a fistulous canal, leading up through much cicatricial tissue toward the gall-bladder, gave vent in the course of several months to great quantities of similar biliary concretions and gravel, but she finally left the hospital much improved, except that she still looked jaundiced. These concretions could hardly be called gall-stones, for they rather had the characters of gall-duct formations, whence it is probable that the abdominal abscess originated in the common or hepatic duct. Her illness, therefore, might be attributed to an attack of catarrhal jaundice leading to permanent thickening and stricture of the lower end of the common bile duct, thus causing permanent but variable jaundice, associated with general dilatation of the hepatic ducts, with a tendency to a retention of bile in them and to the deposition of its solid constituents. Gall-stones, on the other hand, contain a large proportion of cholesterine, though cholesterine is present in the bile in very minute proportions, while the concretions found in the ducts generally consist of the solid constituents of the bile with little or no cholesterine. Many years ago he examined a patient in whom there was a contracted gall-bladder with very thick walls. Embedded in these walls were numerous cavities, from a pea to a bean in size, which were evidently dilated mucous crypts, and within each one of these were found large crystals of pure cholesterine. He has been since inclined to think that the bulk of the cholesterine in gall-stones formed in the bladder is derived, not from the bile, but from the secretions discharged from the mucous surface.

In a paper read by Dr. Ord[36] before the British Medical Association, at Brighton, on some of the rarer symptoms produced by gall-stones, he drew attention.—(1) to the common fact of gall-stones being passed without symptoms. At times they are of very large size. Many cases are on record of death from intestinal obstruction or accident caused by them, without previous history of hepatic colic or pain. In these cases the probable explanation is that adhesion takes place between the gall-bladder and an adjoining coil of intestine, and thus by a painless ulceration an easy outlet is afforded for the concretion. Hence in considering the possible causes of any case of intestinal obstruction we are not to exclude impaction of a gall-stone, even though all story of previous biliary colic or jaundice be wanting. (2) Such cases also indicate another very important consideration, viz., that gall-stones may give rise to various kinds of pain and inflammation, and even death, without the occurrence at any time of jaundice, of which he gives two illustrative instances, in one of which a calculus, altogether too large to have passed through the bile-duct, worked its way, after many attacks of pain, vomiting, etc., first into the peritoneum from the gall-bladder, and then into the intestine. (3) Gall-stones producing severe rigors with intermittent pyrexia, which may readily be confounded with attacks of ague. Charcot regards these cases as the result of septic absorption from the seat of irritation in the duct, and analogous to his uroseptic fever, from like irritation of the urinary passages. Ord regards them, however, as reflex nervous phenomena, in which physicians in this country, where this symptom in obstructive jaundice is very frequently observed, will doubtless fully agree. The attacks are characterized by shivering, sometimes quite severe and prolonged, but with less fever, in the editor's experience, than in true ague, and followed by sweating, but differing from ague in their irregular return and by symptoms of hepatic pain followed by jaundice. So often does this rigor, or at least chilliness, occur in cases of biliary calculus that he has long been accustomed to search for its occurrence in the patient's history as one of the items of differential diagnosis between obstructive and catarrhal jaundice. (4) Not so common is the occurrence mentioned by Ord of glycosuria, produced by the irritation of a gall-stone and ceasing with its removal, of which he gives two cases marked by all the characters of severe diabetes until the hepatic irritation, which he regards as leading directly to it, had subsided. We have already alluded to diabetes supposed to be produced by irritation of the solar plexus in affections of the pancreas, and an analogous nervous origin may be surmised for these examples of this affection as well. Ord would also ascribe the occurrence of pneumonia in cases of gall-stone impaction to a reflex origin similar to the intermittent attacks of fever on the one hand, and to diabetes on the other. He finds support to this hypothesis in the alternating character of the pneumonia with the diabetes, the latter disappearing on the development of the other or of the irritative fever. (5) Gall-stones may produce for their first symptoms sharp attacks of hæmorrhage, of which he gives examples, though usually they are either preceded or followed by the usual symptoms of hepatic calculi; and the bleeding may then be ascribed to the traumatism produced by the tearing of the ulcerated tissues on the passage of a ragged gall-stone.

Vomiting of Gall-Stones.—Dr. T. E. Satterthwaite,[37] of New York, reports the case of a man, æt. 64, who, after an attack of hepatic colic for sixteen hours, vomited a number of gritty particles of a brownish-black color, some flat, laminated and soft, others of a hard crystalline appearance,—the solution of some of them in the vomit giving it an inky-colored sediment. When the vomiting set in the pain was relieved, and though the emesis recurred at intervals during the day, from which some dozen more concretions were obtained, the pain did not recur.

Satterthwaite, beside his own, adds a sixth, reported to him by Dr. C. D. Fitzgerald, of Lathrop, Missouri, of a young woman, æt. 20, who began with symptoms of an irregular, intermittent fever and jaundice, with the development of a painful tumor in the region of the gall-bladder, measuring six inches in length. After six weeks she suddenly vomited and threw up six gall-stones, some of them of the size of a filbert. During the first week of vomiting a dozen or more were ejected, with "crops" of some 15 to 20 at a time coming away by the bowels. She seemed to know the precise time when the "crop" would drop out into the bowel, as she would present then symptoms of shock, so that her life seemed endangered. For fully a month these attacks would recur at two or three days intervals, during which, by actual count, she passed over 150 gall-stones, ranging in diameter from a pea to a good-sized hickory nut. She made, however, a good recovery after an illness of about three months. Her mother had previously died of a hepatic abscess which discharged into the peritoneal cavity. To these may be added a seventh case,[14] under the care of Dr. F. M. Pope, of the Leicester Infirmary, England, of a woman, æt. 40, then in the fifth month of pregnancy, who, on the second day of her admission for an attack of hepatic pain, vomited two gall-stones five-eighths of an inch in diameter, and with many facets. The patient was much collapsed, and the next day vomited a few more stones; on the third day a few more, and, the bowels being freely opened, she passed several per anum, one about three-quarters of an inch in diameter. After this she gradually sank, and died on the 24th day after admission. On autopsy the gall-bladder was found imbedded in an abscess, with its walls in a sloughy condition; and an opening half an inch in diameter, with well-defined edges, was present, leading from the gall-bladder into the duodenum three-quarters of an inch below the pylorus. The stomach was healthy, and there was no ulceration from the gall-bladder into it. Dr. Pope says that Murchison, quoting several authors and a few cases of gall-stone vomiting, says that a gall-stone of any size could not pass backward through the pylorus. and describes a necropsy in which a direct communication existed between the gall-bladder and the stomach; but this case proves that his view was erroneous. The immediate cause of death in his patient Dr. Pope ascribes to septic absorption, which the patient was too much exhausted to resist.

Treatment of Gall-Stones by Olive Oil.—The old treatment of biliary obstruction by large doses of olive oil has been receiving frequent favorable notices in medical journals for some years, and Dr. J. F. McGaston,[39] who has proposed the operation of duodeno-cholecystotomy for cases of permanent biliary obstruction, reports two cases in which the olive oil treatment relieved promptly very pronounced symptoms of this trouble. He had diagnosed, in the case of a woman, obstruction of the duct with dilatation of the gall-bladder and a large biliary concretion in it. The patient took a teacupful of olive oil at intervals of three hours, until three were taken, when the stomach refused to take any more. In the same night she had four evacuations of a dark grumous matter, which continued at intervals for 24 hours, with a peculiar disagreeable smell, which he considers *sui generis* and characteristic of retained bile in the bladder. In the mean time the tumor, extending below the ribs on the right side, diminished and receded under the margin of the ribs, the pain and sensitiveness in the epigastrium ceased; but the darkish passages ceasing in the course of a week and constipation supervening, the old symptoms returned, when the olive oil was repeated, with the result of more dark passages, but less in quantity and gradually diminishing in frequency. Her appetite now returned, and she was able thereafter to eat indiscriminately such vegetables and other articles as she desired, but which often before brought on her accustomed painful sensations. The second case was that of a man with jaundice and a tumor on the right of the median line, extending from the false ribs to the umbilicus. He was ordered a pint of olive oil in two doses at intervals of three hours. This brought away copious discharges of dark semi-fluid accumulations, and though no gall-stones were passed, there was a reduction in the tumor, with marked relief of the sufferings of the patient. On a recurrence of his symptoms he took a half pint of oil again, whereupon his jaundice disappeared and he left the hospital quite well.

Dr. J. Touatre,[14] of New Orleans, cured himself of biliary colic gall-stones by taking a 2¼ grain blue pill in the evening, followed 12 hours later by 12 tablespoonfuls of olive oil. A similar dose of oil was taken a quarter of an hour later, when the patient lay down to sleep on his right side. 60 stones were passed, and the patient enjoyed good health for 3 months, when the trouble began again. He repeated the treatment, and 18 more calculi were discharged. Dr. Touatre has since remained well.

ABSCESS OF THE LIVER.

Pathology and Diagnosis.—Dr. Geo. Harley[14] refers to some observations by Cyr,[32] who enumerates the following differential signs to facilitate the diagnosis of the exact seat of a hepatic abscess: (1) when the abscess is located in the front convex part of the liver, he says that there is dyspnœa, in addition to marked pain, radiating toward the chest and shoulder, while there is only exceptionally any jaundice present, whereas, (2) when the suppuration is limited to the central part of the organ, there is more or less absence of local symptoms, either of the liver itself or of the neighboring organs, pleura, lungs, stomach and intestines, and decided jaundice when the abscess is large; while again, if the suppuration be limited to the under concave surface, there is (3) an entire absence of all thoracic symptoms, with, at the same time, marked stomachic troubles, in the form of uncontrollable vomiting, more especially when the lobe of Spigelius happens to be the seat of the abscess. The direction of the pain, too, in this case is different. Instead of its radiating up toward the shoulder, it radiates downward in the direction of the groin.

Dr. Cyr likewise gives an interesting table of statistics of 563 cases of abscess of the liver, collected from various sources, showing the relative frequency of their different modes of termination, the calculated results being as follow: In 55 per cent. the patients died without the abscess either having burst or been opened, 14.9 per cent. were opened by operation, 10.5 per cent. spontaneously burst into the lung, 7 per cent. into the peritoneum, 5.5 per cent. into the pleura, 1.9 per cent. into the colon, 1.6 per cent. into the stomach and duodenum, and .7 per cent. into the bile ducts; while in the whole 563, 3 opened into the vena cava, 2 into the pelvis of the kidney, and 1 into the pericardium.

Cyr impresses strongly on the reader that a jaundiced tint of the skin is by no means necessary to prove the existence of a grave, or even of an incurable form of liver disease; for, as he says, jaundice may not only be totally absent in the case of hydatids as well as of abscesses, destroying almost the whole of the normal hepatic tissues, but a yellow tint of the skin may be almost equally conspicuous by its entire absence in rapidly fatal forms of malignant disease. In fact, his independent observations entirely confirm the opinions published by the editor some years ago, derived from a careful comparison of hospital statistics, which strikingly pointed out the till then unappreciated fact, that ninety-four out of every hundred fatal cases of hepatic cancer are unaccompanied by any distinctly icteric tint, either of skin or conjunctiva. Jaundice, therefore, though a frequent, cannot be regarded as an essential, symptom of grave liver disease.

Dr. Ferron[40] contributes an excellent paper on "Statistics of Abscess of the Liver, treated by Antiseptic Hepatotomy," which formed the subject of a discussion in the Paris Academy of Medicine. Nothing can illustrate better the advance of antiseptic surgery than the contrast between these statistics of operations performed during the past five years and the dicta on the subject by leading authorities ten or fifteen years ago. Follin and Duplay, in their chapter on Abscess of the Liver, say: "A free incision of the abdominal parietes, with the employment of Lister's methods, appears to us to be a very rash proceeding, and far from receiving the unanimous suffrages of the German surgeons themselves." Dr. Moyet, of the Hôtel Dieu, Paris, in 1873, says that "Simple incision in hepatic abscess is the worst of all procedures that can be adopted." Including a successful case of his own, Dr. Ferron collected 47 published operations of the kind from all parts of the world, in which the abscesses were opened by free incision, with antiseptic precautions, washed out with carbolic acid and drained under antiseptic covering, with 37 recoveries and 10 deaths. Of these 10 deaths, 5 had multiple abscesses, which sufficiently accounts for the result. Of the 5 remaining, 1 was in bad general condition and on autopsy showed a collapsed lung, with an embolus in the pulmonary artery. In 3 others the cause of death was independent of the hepatic abscess, and occurred after its recovery seemed assured, by pleurisy in one, pneumonia from imprudent exposure in another, and diphtheria in the third. The last died from peritonitis on the fifteenth day after the operation, following upon a counter-opening. Dividing, therefore, these statistics into two series, it may be said that: (1) In 39 cases of hepatotomy for single abscess the antiseptic operation was followed by death in only 2.6 per cent. (2) In 8 cases of operation on multiple abscess of the liver, the operation was in 5 cases inoperative toward averting the progress of the disease. This is certainly a very different showing from Dr. Cliquot's report from the register of the Hospital of Gossa, where of 14 cases of liver abscess treated there since 1882, 13 died, confirming the affirmation of d'Hostel that "abscess of the liver inevitably kills." Dr. Rochard, who with M. Perrin was appointed a committee by the Academy to report on this paper of Dr. Ferron's, fully concurred in the latter's positions as to the treatment of hepatic abscesses, with which French surgeons have become much more familiar since the expeditions to Tonquin, Madagascar and Senegal, and M. Trélat avowed himself in absolute accord with him, remarking that the operation in question was performed every day in the hospitals of Paris. "Abscesses of the liver," said M. Trélat, "might be opened if there were no adhesions, if care were taken to unite the edges of the wound in the liver with the edges of the skin wound." The danger of simple puncture arose from the fact that it was not aseptic.

The editor has evacuated three liver abscesses in patients at Bellevue Hospital, with rapid cure in each case, without washing out the cavity at all. The procedure was first to dip oakum into a saturated solution, in boiling water, of salicylic acid and borax. This oakum, when dried, feels silky from the deposition on its fibres of the crystals of these antiseptics. After cleansing the skin and making the incision, a drainage-tube was rapidly inserted and the pus allowed to flow out into the oakum until the abscess was emptied, when a fresh layer was laid on, and over this a cloth dipped in a solution of 1–20 of carbolic acid. If the salicylic acid prove irritating to the lips of the wound, they may be protected by collodion.

Dr. E. Souchon[41] lays down the following rules for performing aspiration in abscess of the liver: (1) When a patient is affected with any disease which may produce abscess of the liver, we should constantly examine the hepatic region. (2) We should explore with the aspirator upon the least suspicion, because the smaller the abscess the greater the chance of success. (3) Acute abscesses when small may be cured by aspiration once or twice; but large abscesses which refill rapidly, after a first aspiration, should not be reaspirated, but should be treated by other means at once. (4) Subacute and chronic abscesses are often cured by aspiration. Success is in inverse proportion to their size. They should be aspirated three or four times before using other measures.

HYDATID CYSTS OF THE LIVER.

The treatment of hepatic hydatid cysts very naturally occupies a prominent place in medical literature at present, in accordance with the change in all forms of abdominal surgery, which the progress of antisepsis has occasioned. This fact is especially noticeable in the French journals, as regards operations on the liver for hydatid cysts, as it is probable that there are more pet dogs distributing hydatids in good society in France than in any other country in the world. A good résumé of the surgery of hepatic cysts, according to the new methods, is given by Marcel Baudouin,[19] from which we extract the following:—

The method introduced by Récamier of inducing adhesion of the cyst to the abdominal wall by the employment of caustics has become superseded by the use of antiseptics, which allows of the liver to be treated as other abdominal organs. Two new methods are here noticed: (1) Volkmann's and (2) that of Lindemann, London. Volkmann communicated his new method at the Sixth Congress of German Surgeons in 1877. This operation consists of two stages: (1) the incision into the abdominal walls; (2) the opening of the cyst, which is delayed for an interval of eight days. This operation of Volkmann's has been almost entirely abandoned in favor of Lindemann's proceeding. Several reasons may be given for the change. It has been established that the peritoneum may be opened and abdominal tumors removed without much danger. This led Lindemann to propose the plan of a large opening into the abdomen and into the hydatid cyst at the same time. At the Ninth Congress of German Surgeons this method was emphatically accepted. In England, Mr. Lawson Tait, in 1880, was the first to announce the employment of large incisions, and his example has been much followed. In France, M. Terrier communicated reports in 1885, and many other French surgeons have reported since then cases of Lindemann's operation. The proceeding of opening by a single large incision presents a notable superiority over the old methods of operating. The situation of hepatic cysts, it is here pointed out, may be *antero-inferior*, originating in the anterior part of the organ and causing prominence on the surface of the abdomen; and *postero-superior*, developed on the pleural surface, and which may press up the diaphragm.

There are, furthermore, cysts which are developed entirely within the substance of the organ. The first form is one in which laparotomy should be performed, and the cyst opened and emptied or removed altogether. The usual antiseptic precautions having been taken, as in ovariotomy, the successive steps of the operation must be guided by the relations of the cyst. When the cyst is intrahepatic, incision of the liver itself will be required, an operation which experience has shown is attended with less danger than might be apprehended. Hæmorrhage is not grave, because the surrounding hepatic structure is sclerosed. The advantages of the operation by large opening of the cysts are, that (1) there is no suppuration of the sac following; (2) if the cyst be pediculated it can be wholly removed; (3) subsequent peritonitis can be avoided. Statistics show a favorable percentage of mortality after the operation, viz., only 7 per cent., which it is believed will be reduced still lower.

The relative fatality of hydatid cysts of the liver, when left to burst spontaneously, has been statistically tabulated by Dr. Cyr, in the work above referred to, in the following order:—

In cysts bursting into the	peritoneum, .	90 per cent. are fatal.
" " " "	pleura, . .	80 " " "
" " " "	bile ducts, .	70 " " "
" " " "	bronchial tubes,	57 " " "
" " " "	stomach, . .	40 " " "
" " " "	intestines, .	16 " " "
" " " through	abdominal walls,	10 " " "

He is strongly in favor of early puncturing and evacuating the contents of the cyst, as being at one and the same time the easiest and speediest means of effecting a cure.

Dr. Arthur Sennett,[14] of Hamilton, Australia, states that he has operated by free incision on a hundred cases with a loss of 10 per cent., but for a year he has given it up for a method which he thinks more efficacious and safer. This affection is very common among men who live in the Australian bush and have bad water to drink; but he was struck with the fact that in fully one-third of the necropsies which he had made in the Hamilton Hospital on such patients, fully one-third presented a shrunken cyst from spontaneous cure. His plan now is to puncture the cyst with a large hypodermic syringe with two needles, drawing off two drachms of the fluid and then injecting a similar quantity of a weak solution, 2 grs. to the pint, of corrosive sublimate. The result in each case has been satisfactory; no suppuration has resulted, and the cyst has gradually contracted. The action of the mercurial is sufficient to destroy the vitality of the parasite, which is never great, and the spontaneous method of cure is thus imitated as nearly as possible. It is well known that an outbreak of urticaria not uncommonly occurs in patients a few hours after puncturing or tapping a hepatic hydatid cyst, and occasionally symptoms of a most urgent kind of extreme collapse, in a few recorded cases ending fatally, have developed after the simplest withdrawal, by a hypodermic needle, of the hydatid fluid. Such a case is reported by Dr. L. Humphry,[14] and, to test the theory which has been offered that hydatid fluid contains some soluble poison similar to that of urticaria-producing shell-fish, Dr. Humphry requested Prof. Roy to experiment on animals with some of the hydatid fluid obtained from his patient. One injection of 20 c.c. into the jugular of a large dog resulted in slowing the heart-beat from 70 to 33 per minute, and the respiration from 37 to 17, with a fall of blood pressure from 137 mm. to 63 mm. (hydrarg.). As the animal appeared to be dying, a small dose of atropine was injected into its vein, which was followed by a gradual but continual rise of blood pressure until it remained at about 125 mm., although during the rise 40 cc. of hydatid fluid were injected at intervals of about five minutes. The pulse rose to 72 and respiration to 28. The animal was then killed, and the chief characteristics of the necropsy were the enormous congestion of the liver and the free flow of urine from the cut surfaces of the kidneys. The marked change in the symptoms of hydatid poisoning caused by the atropine seems of much interest, as well as of practical importance, for future cases of such accidents in withdrawing hydatid fluid in the human subject. In the post-mortem account of a fatal case of the kind recorded by Mr. Bryant, it was found that immediately within the hydatid capsule the trocar had wounded a large vein, and it was supposed that after the withdrawal of the trocar some of the hydatid fluid entered the wounded vessel.

HYPERACID VOMITING IN TABES.

Prof. Rosenthal,[8] of Vienna, has a paper on hyperacid vomiting in tabes and the reaction of the urine in most cases. In the obscurity of our knowledge of the gastric processes and of their relation to the nervous system, he considers that the vomitive forms of gastric crises in tabes accompanied by hyperacidity and hypersecretion are worthy of note, because clinical symptoms are here connected with certain determinate post-mortem lesions. In some cases of tabes the gastric crises are ushered in by lancinating neuralgic and cardialgic pains and palpitations. The vomiting sometimes lasts for days, and, as Rosenthal has determined, contains from .30 to .32 per cent. of HCl. In his cases the acidity remained in a high degree even after fifteen hours' fast, and this hyperacid secretion always went along with the symptoms of spinal irritation and subsided with it. The anatomical basis for the cardialgial and hypersecretive vomiting may be found in demonstrable degenerations in the territory of the vagus centres; for a subependymal sclerosis attacks the posterior vagus nuclei which are found in the bottom of the fourth ventricle, and as these cells are in connection with the sensory fibres of the vagus, the initial cardialgia is thus brought about, and then by further spreading of the irritation the vomiting is produced. The increased secretion of a fluid containing HCl must therefore be caused by the excitement of determinate secretory nuclear cells; the cardiac and pulmonary nuclei being also involved, the weakening and quickening of the heart's action, as well as the labored breathing, become thus intelligible. That the gastric crises and vomiting may sometimes be the first symptoms of the onset of tabes, was shown in a case seen by the editor in March, 1887, in consultation with Drs. S. Fleet Spier and T. C. Clark, of Brooklyn, N. Y. The patient, a young gentleman, suffered from intermittent attacks of uncontrollable, very acid vomiting, which would sometimes last for weeks, and yet the usual signs of gastritis were not present. The suspicion then entertained that his vomiting was due to a beginning tabes was after some months fully confirmed by the development of the characteristic lancinating pains and motor disorders of locomotor ataxia.

Many cases of hysterical vomiting are also to be classed with the medullary forms of hypersecretory vomiting. They are introduced by cardialgia, and often when the patient has fasted, great quantities of slimy fluid, containing free acid, are ejected. The frequent combination in such cases of slow pulse, dyspnœa and cough, point to irritation of the bulbar vagus centres. For this form of acid vomiting he recommends alkaline waters with bismuth and morphia. In this connection the editor may mention two cases of vomiting of intensely acid matters in two female patients, each under 30 years of age, and which exceeded in severity any cases of vomiting that he ever had to deal with. Both were of highly neurotic families, the first having an insane sister and a dipsomaniac brother; her mother died also in an epileptic fit. The second has a very extensive history of nervous disease on her mother's side, including three cases of paralysis agitans, and one, an uncle, died of bulbar paralysis. The patient herself is greatly troubled with attacks of pruritus. After every other means recommended for the arrest of vomiting had been unavailingly tried in each case, they both had the affection promptly checked by hypodermatic injections of morphia, which, however, had to be repeated in full doses in one case for two days, in the other for five.

Prof. Tumas[14] contributes a series of researches on the vomiting centre. By touching different parts of the floor of the fourth ventricle in dogs with a weak solution of apomorphia so as to induce vomiting, he was able to localize with tolerable precision the situation and extent of the vomiting centre, which, he says, lies in a space about 5 mm. in length by 2 mm. in breadth before and behind the columns and in the deeper layers of the medulla.

CIRRHOSIS OF THE LIVER.

Cyr,[32] in his work recently issued, considers that a chronic atrophying condition of the liver is always ushered in by dyspeptic symptoms, a loss of appetite and distaste for food, accompanied by thirst. Morning sickness is also not uncommon, and the efforts of vomiting are occasionally distressing, the ejected matters being either sour or bitter, according to the proportion of gastric juice or bile mixed with the phlegm in them, which is usually very abundant. He also calls attention to the frequency with which hæmorrhages are met in advanced cases of cirrhosed liver,—even fatal hæmorrhages. From his calculations, bleedings appear to occur in the proportion of thirty-eight in the atrophic and forty in hypertrophic cirrhosis. Although he thinks complete cure is rare in cases of chronically hardened liver, yet he admits that cases of recovery are much more frequently met with nowadays than formerly; and this he attributes mainly to the fact that within the last few years it has become the fashion to entirely abandon the use of alcoholic drinks during their treatment. In the early stage of cirrhosis he strongly advocates the administration of alkaline mineral waters, and that of Vichy in particular. He also recommends, combined with employment of drugs, the free use of local counter-irritation, even going so far as to counsel the use of the cautery. One of the worst signs that can manifest itself in a case of cirrhosis is the augmentation of jaundice, which is not infrequently accompanied by febrile symptoms and an increase of ascites, both of which are unfavorable complications. To the cirrhosis resulting from syphilis he gives special attention, regarding it, as most authors do, as a most dangerous form of liver affection, even when unaccompanied with either jaundice or ascites. For treatment of this form, mercury and potassium iodide, as usual, are our chief resource.

Malarial Cirrhosis.—Dr. Lancereoux, of the Hôpital de la Pitié, of Paris, delivered some able lectures on malarial cirrhosis of the liver, which, besides clearly demonstrating its specific form and characters, are valuable for distinguishing the different varieties of cirrhotic changes of this organ, as at present established. From an etiological point of view, Lancereoux recognizes three forms only of primary cirrhosis,—(1) as a result of alcoholism; (2) as a result of malarial infection; and (3) of syphilis. All other varieties of cirrhosis are but secondary changes, depending upon other primary disorders, such as passive hyperæmia of the liver from heart disease, from bile obstruction, or from cancerous infiltration, with specific histological changes characteristic of each. In giving the characteristic signs of malarial cirrhosis he says that it is distinguished clinically,—(1) By a history of chronic malarial infection. (2) By a great enlargement of the liver, and of the spleen also. (3) By a persistent and exceptionally chronic jaundice, lasting in some cases from five to seven years, and which is further characterized by a dark-brown discoloration of the skin, and not by the greenish-yellow jaundice from impaction. It is readily distinguished from the common alcoholic atrophic kind by the great enlargement of the liver, by the absence of dropsy and of distended abdominal veins, and by the implication of the spleen. It is distinguished from the alcoholic fatty hypertrophic cirrhosis by the edges of the liver being sharp and well defined on palpation, whereas the fatty cirrhosis presents a roughened and rounded outline of the boundaries of the organ. The distinction is at first not so easily made out in carcinoma with jaundice, but the chronic history of the malarial and the development after a time, in the cancerous case, of nodosities, serves to settle the diagnosis. The absence of any cause of amyloid degeneration (phthisis, chronic suppuration, etc.) will serve to exclude that form of hepatic or splenic enlargement, while the blood changes and the icterus will readily exclude leukæmia.

Among the symptoms of malarial cirrhosis, epistaxis is common, but not to the same extent as bleeding of the gums or of the intestines. There are, however, a good many dyspeptic derangements, with vomiting and temporary diarrhœa. The respiratory organs are unaffected, but heart murmurs are at times present at the right border of the sternum, due, as Potain pointed out, to tricuspid regurgitation. The skin is rough, and often there is troublesome pruritus. One of the rarer, yet characteristic, symptoms, is hemeralopia, which is intermittent or variable,—of which he cites examples, and which is to be attributed to pigmentary deposits in the retina. Scarpa long ago noticed this symptom among peasants who lived near the rice-fields of Pavia, and it develops only in chronic cases. The patients who succumb to malarial cirrhosis present finally, after a long history, all the usual symptoms of failure of hepatic function. They progressively grow emaciated and weak, with loss of appetite and œdema of the legs, with a little ascites. Fever, which has been long absent, now returns, but has more the form of attacks of so-called "liver" fever, which accompanies the severe forms of icterus. The tongue becomes rough and fissured, delirium and hæmorrhages occur, and they die comatose. On autopsy the liver is found greatly enlarged and doubled in weight, without its shape, however, being lost. Its surface is very smooth, without prominences. The consistency of the liver tissue is increased to a degree between the hard alcoholic atrophic and the alcoholic fatty liver. The liver tissue does not grate under the knife, nor has it the elasticity of the atrophic form, and the section shows a variable coloration from the abundance of pigmentary and biliary corpuscles, with the same granulations as its superficies; but they do not project above the cut surface, nor tend to enucleation like the large papillæ of common cirrhosis. The proliferation of the connective tissue is not found to accompany the venous radicles of the portal system, nor the perihepatic net-work of vessels, which nowhere show any trace of endophlebitis. This explains the absence of dropsy dependent upon portal obstruction. Many capillaries are seen to traverse the new connective tissue, so that the circulation is everywhere free. The arrangement of the connective tissue, while not, properly speaking, similar, yet is not so specially distinctive of this form of cirrhosis as the consistency of the tissue itself, which in fact is rather embryonic in its character, and is organized only in the interlobular spaces, but nowhere developed into the perfectly formed, fibrous, rigid and unyielding bands of common cirrhosis. The new growth only presses on the borders of the lobuli, which therefore appear fringed and irregular, but it does not invade the lobule itself or implicate its central vein. The biliary canaliculi are extensively distended by this proliferation, as they are in many cases of common cirrhosis, and the extent of this interference with their function leads to their becoming very prominent on the one hand, and to a bile-stasis on the other, which causes the chronic jaundice of the disease. The larger ducts and the common duct are always free. The liver cells on the surface of the lobuli are compressed and elongated, and their protoplasm diminished; in the centre of the lobule they are of normal form and only filled with bile pigment and fat granules. Dark pigment granules abound, and are characteristic of malarial cirrhosis; but they are limited to the neighborhood of the vessels in the stroma, and seldom are found in the liver cells themselves.

As to treatment, in the first stage of malarial cirrhosis, the measures should be the same as in malarial congestive liver disorders,—quinine and hydropathy affording the best means to combat the condition. In the developed stage, hydropathy is still indicated; but when accomplished, the potassium iodide is to take the place of quinine, and to be continued through months and years, along with a rigidly exclusive milk diet. In a child of eleven years in whom the liver and spleen were both greatly enlarged, these measures, kept up for ten months, caused these organs finally to return to their normal dimensions.

Artificial Cirrhosis.—At a meeting of the Society of Biology, Strauss[15] communicated the results of experiments which he had made with his assistant, Berne, on the artificial production of alcoholic cirrhosis of the liver in animals. Dogs being found too sensitive, the experiments were conducted on rabbits, in whom he introduced daily, by means of an œsophageal tube, half an ounce of absolute alcohol with methyl alcohol, diluted with three parts of water. Immediately after receiving this injection the greater part of these animals fell as if paralyzed, and for several hours they lay in deep coma. When this dosing was continued for some months and the animals were killed, the experimenters invariably found the usual lesions of alcoholic gastritis,—thickening of the mucous membrane, ecchymotic petechiæ of the surface, etc.; but what especially attracted their attention was the pathological condition of the liver. This organ did not present to the naked eye any marked alterations; it was smooth on surface as well as on section; the acini, nevertheless, were surrounded by a reddish gray line, and in animals which had been kept most of the time intoxicated for three or four months, the ultimate perilobular portal spaces were found infiltrated with embryonic cells. In hares which had been constantly subjected to the poison for seven or eight months, the hepatic lobules were completely surrounded by a crown of connective tissue cells, and presented typical forms of annular, perilobular and monolobular cirrhosis. "Nothing," says M. Strauss, "is more natural than this localization. The alcohol coming to the liver by the finer branches of the portal vein, might be expected first of all to exert a baneful action in points where its contact is most intimate." The hepatic system of veins (intralobular, sublobular and hepatic trunks) never, according to Strauss' researches, participates in the process of alcoholic cirrhosis.

Cirrhosis in Children.—Professor R. Palmer Howard,[44] of Montreal, read a paper before the Association of American Physicians, at Washington, on this comparatively rare affection in children, based on 2 cases in his practice, and 61 recorded in medical literature. Considering the causative relations of the cases analyzed, we find that in 10 the children were addicted to the use of spirits; in 7 there was a clear history of syphilis; in 7, perhaps 8, cases, tuberculous disease coexisted; but in more than one-half the cases the etiology was obscure. It is possible, as Botkin originally suggested, that the infectious diseases may originate chronic inflammatory changes in the organs, in proof of which may be cited Klein's discovery of an acute interstitial hepatitis in 8 cases of scarlet fever. But in only 9 of the 38 cases, which cannot be referred to in any of the established causes, had acute infectious disease preceded by a longer or shorter interval the cirrhosis. For the remaining cases Howard is inclined to favor Budd's suggestion,—that the products of faulty digestion and certain stimulating kinds of food may incite interstitial hepatitis. Among such products the ptomaines may be important, and it is possible that they may be as irritating or even more so than alcohol. In certain children a rich, stimulating diet may act as injuriously as alcohol on an organ constitutionally prone to interstitial change. That such a tissue weakness may exist is suggested by its occasional occurrence, as in Howard's cases, in members of the same family.

Of the character of the cirrhosis in the 50 non-syphilitic cases, in 19 the organ was atrophic, in 13 hypertrophic, in 6 of normal size, and in 16 this point was not mentioned. The symptoms are practically the same with those of cirrhosis in adults. In the discussion of this paper Dr. Wm. H. Welch, of Baltimore, reported the case of a child twelve years of age, who had come from the coast of Africa and suffered with malaria. Both liver and spleen were deeply pigmented.

Dr. Wm. Pepper, of Philadelphia, reported a case in which cirrhosis followed measles in a child eight years of age. There was no syphilitic history. During the attack of measles there were symptoms of hepatic disorder, as shown by occasional attacks of catarrhal jaundice. Subsequently the symptoms of developing cirrhosis made their appearance, and death in a comatose condition supervened. At the autopsy a typical hob-nail liver was found.

Treatment.—Discussion has appeared in Continental journals as to the curability of cirrhosis. At a meeting of the Paris Société des Hôpitaux, M. Troisier[4] communicated a case in which the ascites from cirrhosis had disappeared under the influence of potassium iodide and strychnia, and the visceral lesion itself had seemed to be cured. He asks whether there does not exist a particular form of alcoholic cirrhosis in which the lesion, stopping short of the destruction of the liver cells, is susceptible of retrogression, and he relates another case in support of this hypothesis. A confirmed drinker, æt. 68, was under the care of a colleague, and presented all the symptoms of hepatic cirrhosis. Between December and September, 165 litres of fluid were removed by tapping at different times. At the present time the peritoneal cavity is empty, the liver slightly enlarged, and the patient in excellent health.

Dr. Schreph,[33] Vienna, gives a case of cirrhosis hypertrophia cured by calomel. A woman, æt. 28, had all the symptoms of the disease well marked. The liver was much enlarged. After a course of Carlsbad had been tried for five weeks in vain, and strong purges followed by free operations had afforded only slight relief, the author, prompted by the suggestion of Prof. Nothnagel, ordered an absolute milk diet and gave $\frac{6}{10}$ grain of calomel three to five times daily. On the second day of this treatment the fæces had already assumed a yellow color and the urine had lost most of its high coloring, and in a few weeks icterus and ascites had disappeared. The liver was much smaller, and menstruation had again set in. In three months the patient had taken 100 doses of calomel and complete recovery had resulted.

We may add that all physicians of much hospital or general experience can supplement such histories with cases of their own; but the instances cited show that recovery can be hoped for only when enlargement of the liver is present. Therefore it is probable that engorgement rather than cirrhosis had most to do with the production of the ascites and of the other symptoms. The early resort to tapping is much to be recommended, for the editor has repeatedly noticed the progress of the patient's decline arrested, for a time at least, with its employment, if no ultimate cure resulted. The tincture of the chloride of iron with strychnia has seemed to him to be beneficial, as well as the iodides and the corrosive sublimate. Cyr's recommendations for the treatment of hepatic congestion seem worthy of employment also, viz.: (1) Application over the liver of compresses of cold water often renewed, and in acute cases two or three leeches about the anus. (2) At evening ¾ of a grain of calomel should be taken, followed the next morning by 5 drachms of Glauber salts. (3) A beverage of milk and Vichy waters, or 75 grains of ammonium chloride in a quart of water. (4) A douche to the hepatic region, while the patient is reclining, of water at a pleasant temperature. The editor may add that in simple engorgement with jaundice he has repeatedly seen good effects from faradization for twenty minutes of the region of the liver, one pole being placed at the middle dorsal intestine and the other passed constantly over the right and left lobes, with occasional pause over the gall-bladder.

Surgeon-General W. Stewart,[14] of the East India Service, speaks again very strongly of the place which chloride of ammonium has as a remedy in tropical congestion of the liver. He lays stress upon the special and characteristic symptoms produced by the drug in hyperæmia of the liver, which are peculiarly and directly referable to its action on the organ, and for which physicians should be on the look-out, lest they be mistaken as evidences of the medicine disagreeing; for on the contrary they are signs of its successful action, and as such may serve for diagnosis, as they do not develop without hepatic disorder be present. These symptoms occur shortly after the medicine is taken, in from five minutes to half an hour. Sometimes a shock is felt, or as if something gave way in the side; at other times a succession of shocks is felt in the hepatic region, accompanied or not by a pricking sensation, or as if cold water were trickling down the side, or described as a "pulling" from one hypochondrium to the other; or "clawing" or "gnawing" sensations through the liver are mentioned. For conducting this treatment with the ammonium chloride, the patient should remain strictly in bed during the whole course. He should take only small quantities of milk and farinaceous food, barley-water being taken freely as a drink. Diarrhœa does not contra-indicate the chloride, for it is itself the best remedy for looseness of the bowels dependent upon hepatic congestion. The only contra-indication in acute cases is high fever with dry skin, when the ammonium acetate should be given with diaphoretics, and then as soon as the pyrexia subsides the treatment should be begun, twenty grains of the chloride being given twice or thrice daily till the hepatic symptoms subside.

CONGESTIVE HYPERTROPHY.

Puncture of the Capsule of the Liver for Congestive Hypertrophy.—Dr. George Harley,[34] of London, thinks that in this condition, however induced, whether by alcoholism or malarial infection, etc., the usual treatment by counter-irritation, cupping or blistering does often more harm than good, and that the employment of hot fomentations and saline purgatives promises better. But considering that nearly all the pain and discomfort in hepatic congestive hypertrophy is directly due to the pressure of the hepatic tissues, from being confined within a strong, inelastic, fibrous capsule, and knowing the immediate relief which follows upon the puncturing of other equally unyielding fibrous coverings when their contents are in a state of inflammation, he determined to try puncturing the capsule of Glisson, for just such conditions as this procedure benefits in orchitis by puncturing the tunica albuginea. He was also led to this by noting the comfort many patients experienced after an unsuccessful exploration of the liver for an abscess than they did before the operation. Dr. Harley then gives cases in which he has had all the success he anticipated from a resort to this operation. One, a severe case of hardened hypertrophy of the liver in a gentleman æt. 52, who was accustomed to drink a bottle or a bottle and a half of champagne with his dinner, while living in South America, and who had tried a course of six weeks at Carlsbad without effect, was punctured by him at the same sitting with a No. 2 English sized trocar twice in the left, and once with a No. 5 trocar in the right lobe, and then the canula was retained for a few minutes in the wound. The relief felt was immediate, and in 18 hours the doctor found him sitting up, busy writing letters, and complaining only of a little tenderness at the site of the punctures. In three days he walked a quarter of a mile to the doctor's office, and stated that he felt perfectly well, so that he wished to start at once for South America again. The operation, as indicated, consists merely of puncturing from three to six times, according to the gravity of the case or the strength of the patient, the anterior surface of the liver, with trocars from Nos. 2 to 6, and leaving the canulæ in for a short time to permit of the oozing away of any liquid that may chance to be at the seats of the puncture. No anæsthetic is necessary, nor any other precaution beyond using clean instruments, lubricated with carbolized oil, and then putting 2 inch sized pieces of diachylon plaster over each puncture, and applying an abdominal bandage to keep the abdominal parietes well against the liver.

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DISEASES OF THE INTESTINES AND PERITONEUM.

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DYSENTERY.

Etiology.—The cause of dysentery is still one of the vexed questions of the age and the various observations of the year past continue to draw attention to meteorological changes, malaria, decomposing animal, vegetable, and fæcal matters as factors in generating it.

A study of 2507 cases treated in the St. Wladimir Children's Hospital,[1] in Moscow, during five years, and a comparison with the meteorological changes show that continued elevation of temperature increases the number of cases, especially in the first half of summer. Humidity of the air adds to the pernicious influence of extreme heat or a sudden cooling of the air. Barometrical pressure, rainfall, and thunder-storms have no influence.

The experience of careful observers in malarial districts still confutes the general opinion that malaria and dysentery have a common origin. Ghent,[2] in a practice of thirty years in a malarial atmosphere, is satisfied that the two diseases, though coincident in the same season or in the same individual, are distinct as to cause. Malarial infection may predispose to the influences which produce dysentery, but the causes are not identical.

As usual the testimony as to the influence of impure drinking water is contradictory. Bettinger,[3] who was with the French Army in Tonquin, where the most intense form of dysentery prevailed, did not believe that the drinking of the foul water of the marshes induced the disease. It did not happen to some who drank this water, and officers fell victims to it who drank water deprived of all organic matter.

Is dysentery caused by a specific germ? Kartules,[4] of Alexandria, found an amœba in the discharges of 150 typical cases of dysentery which he believes to be the constant micro-organism of the disease. It is not found in ordinary diarrhœa. It is seen in the stools, the intestinal contents, and in the intestinal walls. Hlava found an amœbiform large-grained protoplasm in the stools in 65 cases, and in the intestinal mucous membrane and submucous tissue in 20 cases. Kartules cultivated the amœba, but did not succeed in producing the disease in animals by inoculation, and Hlava also failed to infect animals by the injection of the fresh stools of dysenteric patients into the rectum and duodenum of animals. Only a transitory catarrh followed.[5] So far this testimony shows that micro-organisms found in dysenteric stools are not the cause of dysentery. No etiological or anatomical distinctions have yet been found between epidemic and sporadic dysentery. In support of the infection theory Briggs[6] gives the history of an epidemic of dysentery in which a cessation of the disease followed upon a disinfection of the closets and beds.

Pathological Anatomy.—In a case of dysentery an abscess of the liver opened below the spleen, forming a large collection of pus behind the peritoneum and around the left kidney. The pus made its way to the left iliac fossa and the abscess was about to be opened here when the patient died.

Treatment.—Popular favor in India still holds to the ipecacuanha treatment of dysentery. McDowell,[8] after a service of thirty-five years with the British Army, says that the horrors of dysentery are disappearing since this treatment has been employed. He gives a preparatory dose of 20 drops of laudanum one hour beforehand, and orders a mustard plaster over the stomach. 20 to 25 grains of ipecacuanha are given at bed-time. This treatment is repeated every evening, and no fluid is to be swallowed afterward. By the third day the blood, mucus, and pain have generally disappeared. The experience of those who have had most to do with epidemic dysentery in this country has led to a division of favor between the specific treatment by ipecacuanha alone, and purgation by salines. A supposed portal congestion furnishes the theory for the purgative plan of treatment, although its good effects are confirmed by experience. The fact that the supporters of purgation advise the constant use of opiates afterward, and that those who recommend ipecacuanha do not find it necessary to have recourse to them, gives ground for belief that the latter is the more successful method.[9]

Among the remedies suggested is the tincture of cannabis indica in doses of 15 to 20 minims three times daily; but the suggestion is based upon a limited number of cases, and mention is made of the drug to show how new remedies creep into popular notice on the most limited basis of experience.[10]

Antiseptic remedies by the mouth are advised in dysentery. Among these salol, which is said to pass unaltered through the stomach and is changed into salicylic acid in the intestine, has been tried, and it is said with success. Other antiseptics have been given internally and could be employed profitably in connection with rectal irrigation and antisepsis. (Goelet.[11])

Treatment by the rectum offers the most rational means of cure, and experience is adding to the already large amount of evidence in its favor. To simple irrigation with hot or cold water, antiseptic irrigation has been added, and there is much hope that in this lies the future of the successful treatment of dysentery. Fordyce[12] washed out the rectum with water as hot as could be borne and then irrigated with one quart of a 1–10000 solution of bichloride of mercury. Pain and tenesmus were at once relieved. A one-grain opium suppository was then introduced and the patient slept for seven hours. In twelve hours the same treatment was repeated. The patient was well in six days. Repeated trials in other cases confirmed this experience. Hot water irrigation (105° to 115° F.) divides favor with ice water enemata. The latter is said to give great relief in dysentery with high fever and great tenesmus in children. There is no question that irrigation and antisepsis combined by cleansing ulcerating surfaces, washing away the débris of necrosing tissue and destroying all micro-organisms, will directly reach the desired end. The defect lies in the impossibility of reaching the diseased surface in the descending and transverse colon. This mechanical difficulty overcome, the method must be successful.[13]

Theoretically cocaine would be expected to be the remedy in tenesmus, and some testimony in its favor has been given. It can be administered in enema or suppository, but its effects are temporary and disappointing.

DIARRHŒA.

Acute Catarrh of the Intestines.—Illingworth has used injections of carbolized water in diarrhœa, and writes that the effect is marked. One ounce of a one to twenty carbolic lotion is added to a teacupful of warm water, injected into the bowel and retained. In catarrh of the duodenum and bile ducts with icterus, cold water injections have been used with advantage by Krauss,[14] of Vienna. Two to four pints of water at a temperature of 59° to 72° F. are thrown into the rectum; the treatment is repeated four or five times. This method quieted intestinal and gastric irritation, relieved nausea, and the fæces soon showed bile. Chauffard[15] reports seven cases in which the obstruction to the bile flow was overcome in from four to six days by injections of from one to two quarts of cold water. Active peristalsis, sometimes with colic, followed. The result is supposed to follow from reflex contraction of the gall bladder and large bile ducts.

Chronic Catarrh of the Intestines.—The successful treatment of chronic catarrh is a treatment by diet; drugs are unimportant, unnecessary and frequently injurious. Above all things, milk in some form is the starting point in all cases. In the chronic diarrhœa of hot countries, which is so general and so rebellious, this question of dietetic treatment has received diffuse attention by French authors. In a long paper by Drs. Bertrand and Fontan[16] the subject is elaborated in all its details. They give the following useful rules for diet: (1) Liquid stools: milk, pure or peptonized, with or without meat peptones; bouillon or beef tea, if there is an intolerance of milk. (2) Soft homogeneous stools: milk, rice with milk, one or two eggs. (3) Moulded stools: milk, rice, eggs, beef juice, boiled fish, white meat of poultry; later, milk, eggs, beefsteak broiled, toast; later, broiled steak, roast beef or mutton, starchy, and later, green vegetables; wine, beer.

De Champeaux[17] reports favorably the use of sulpho-carbonated water, five to fifteen drachms in milk during 24 hours. It is supposed to act by destroying micro-organisms. The formula is: bisulphite of carbon, six and one quarter drachms; water, one pint; essence of mint, thirty drops. Place in a flask containing three pints and shake thoroughly. Bichloride of mercury has been found to succeed in the typical, "white," or "hill" diarrhœa of India by Macpherson,[18] in cases where other drugs had failed.

For further reference to this subject see *Intestinal Antiseptis.*

Pancreatin was employed in tropical chronic entero-colitis by Bertrand.[19] It was given without other medicament, and was immediately followed by beneficial effect in doses of 5 to 15 grains taken with milk. 15 grains in the course of the day was sufficient to produce the desired result. Among old remedies revived, oxide of zinc appears prominently. The extravagant statements made in its support, as by Dupré,[20] do not differ from the claims made many years ago and are equally unworthy of adoption.

MEMBRANOUS ENTERITIS.

Field,[100] in the Fisk Fund Prize Essay, has collected all the reported cases of membranous enteritis, one hundred and thirteen in number, with three hitherto unpublished cases of his own. 80 per cent. were women, and as a rule of nervous temperament and of middle age. The attacks recur periodically in typical cases, and consist of nervous and digestive disturbances followed by the discharge of membranous shreds and moulds of the intestine. These are composed of mucin and not of fibrine or albumin. From the periodicity of the attacks, the nervous complications, and the simultaneous discharge of pseudo-membranes from other organs, and its association with uterine disease, the conclusion is reached that it is a neurosis. Only six fatal cases are reported, death occurring not directly from the membranous disease. The treatment is diet and regulation of the bowels; arsenic has been much praised, and washing out of the colon and injections of mild astringents are recommended. A case of perforation of the duodenum in a case on membranous enteritis is recorded.

SIMPLE ULCER OF THE DUODENUM.

Round or simple ulcer of the duodenum has a well-recognized place in pathology. It is a rare lesion, but the literature of the subject is by no means meagre. Bucquoy[92] has contributed a very full study of the subject and Littlejohn,[93] Coates and Gairdner,[94] Dutil,[95] Wissing and Wallis,[96] and Rothman,[97] have reported cases or exhibited specimens during the year.

Etiology.—Ulcer of the duodenum is more common in adult life and more frequent in men than in women, the opposite being the case with gastric ulcer. Alcohol in excess is supposed to be a cause. Ulcers due to external burns are not of the same nature.

Pathological Anatomy.—Out of 261 cases of ulcer, Wier found 28 in the duodenum. The most common seat of the ulcer is near the pylorus on the anterior wall. There may be one, two, or more. In shape the ulcer is round and infundibuliform. Perforation and peritonitis may accompany it. When the ulcer heals, cicatrization may, by contraction, narrow the pyloric opening and lead to dilatation of the stomach. The fact that the ulcer has its seat in the beginning of the duodenum, above the point where acid reaction changes to alkaline reaction, goes to show that the genesis of duodenal ulcer is the same as that of gastric ulcer. The primary lesion is an arrest of nutrition from embolism or thrombosis with consequent destruction of tissue by acid corrosion. In opposition to the embolic theory of Virchow, Gaillard has shown that the round embryonic cells, collected between the glands and in the substance of the muscular and mucous layers, degenerate into pus cells, and becoming free, leave in their place a small cavity or an erosion which enlarges by destruction of its borders.

Symptomatology.—Duodenal ulcer may remain latent like ulcer of the stomach, the first indication of its existence being often an alarming hæmorrhage or perforation and suddenly developed peritonitis. The only early symptom which may in some cases attract attention is a profound and progressive anæmia. Other symptoms met with are discomfort or violent colics 3 to 4 hours after food is taken, or severe pain and vomiting, pain in the right hypochondrium and sensibility to pressure over the duodenal region to the right of the median line and below the lower edge of the liver. The hæmorrhages are apt to occur during a violent duodenal colic 3 to 4 hours after eating. Blood escapes by vomiting or is passed by the bowel, of a dark color; great prostration, syncope, and acute anæmia result from the hæmorrhages. In one fatal case, in a man in perfect health, reported by Dutil[96] violent colic set in two hours after eating, attended with persistent vomiting and tympanitic distension with constipation and almost fatal suppression of urine. These symptoms lasted 14 hours and were supposed to be due to intestinal strangulation. The patient died 18 hours after the onset of the symptoms. A round ulcer with sharp cut edges was found on the anterior wall of the duodenum just below the pylorus, perforated at its base, with resulting peritonitis.

Diagnosis. — Duodenal ulcer is recognized by sudden and recurring intestinal hæmorrhages occurring in men, with free intervals in which there are no evidences of gastric derangement. This disease has been mistaken for cholera, lead colic, gastric ulcer, intestinal strangulation, etc.

Prognosis.—The prognosis is unfavorable as a rule, but out of Bucquoy's 5 cases, 4 ended in recovery. The progress of the disease is slow, with exacerbations and remissions.

Treatment.—A pure milk diet, or milk diluted with Vichy water, is essential. The same rules applicable to ulcer of the stomach apply here. If a hæmorrhage occurs, ice should be applied over the abdomen and hypodermic injections of ergotin given.

TYPHLITIS; PERITYPHLITIS; PARATYPHLITIS; APPENDICITIS.

Nature.—Judging from the expressions of opinion contained in recent journal articles, much confusion exists in the minds of their authors as to the true pathology of inflammatory conditions in the region of the cæcum and appendix vermiformis; and the question arises whether our opinions are not more the result of preconceived theoretical views than of post-mortem study. Typhlitis (inflammation of the walls of the cæcum); perityphlitis (inflammation of the peritoneum covering the cæcum); paratyphlitis (inflammation of the connective tissue behind the cæcum); and appendicitis (inflammation of the appendix vermiformis), are some of the terms applied to inflammatory states in this region. As regards the first of these diseases (typhlitis) post-mortem examinations make it doubtful whether the walls of the cæcum and the post-cæcal connective tissue are the seat of marked inflammation except in rare instances from impacted fæces and foreign bodies. Perforations from round ulcer and other lesions of the cæcum have been observed by Osler.[21]

The record of autopsies shows that the appendix is almost invariably the seat of the lesion, which is accompanied by symptoms of pain and tenderness in the right iliac region, fever, vomiting, constipation, dorsal decubitus and flexing of the thighs, and by the development of a tumor or the evidences of pus collection and local or general peritonitis. The location of the abscess is in the right iliac fossa, in the right lumbar region, behind the cæcum, or in the pelvis. Within the abscess cavity is the appendix, inflamed, ulcerated, perforated, gangrenous, or destroyed. A foreign body, most frequently a fæcal concretion, is found in the appendix or in the cavity of the abscess.

There is, however, a large class of cases in which the symptoms above detailed are not followed by a fatal termination. In some instances the symptoms, even to the tumor formation, recur more than once: in many they abate under counter-irritant and resolving treatment, sometimes with the discharge of hard fæcal matter, intestinal concretions, or foreign bodies, as gall stones. In these cases it cannot be supposed that ulceration and perforation of the appendix has occurred, and it is here that the theory of typhlitic and paratyphlitic inflammation is adopted in explanation. This theory needs confirmation, although it is so generally accepted and although no better one may be substituted for it.

Etiology.—Cases continue to be recorded of perforating appendicitis caused by foreign bodies as beans, fruit stones, berry seeds, and fæcal concretions, the latter being the most common. Simple catarrhal inflammation leading to ulceration and perforation may begin without the agency of a foreign body; at least no foreign body can be found in some cases.

Pathological Anatomy.—The question of the anatomical seat of the pus collections which follow appendicitis is an important one. The pus is frequently intraperitoneal, contained in circumscribed peritoneal sacs formed by the adhesion of adjacent intestinal loops: this is proved by post-mortem examination and by the operations for the opening of these abscesses. In a case reported by Dr. Albert Wilson and Dr. Goodlee[22] an incision was made below the anterior superior spinous process, the cavity was opened and found to be bounded posteriorly by coils of small intestine held together by soft adhesions: the pus accumulation was localized in this cavity, which was emptied and cleansed. Two days after the operation vomiting and straining at stool caused the protrusion of a knuckle of intestine between the lips of the wound. The bowel was forced back, air and offensive pus escaping from the wound. The patient made a good recovery. The lesion proper of fatal cases is, without doubt, appendicitis with destructive inflammation and perforation of its wall. What the lesion is in non-fatal cases is a matter of doubt. It might be supposed that quite often the cæcum was the seat of the lesion, and that simple inflammatory effusion forms the tumor and undergoes easy resolution. Numerous recoveries are reported of cases with such inflammatory indurations.

Symptomatology.—Jaundice has been seen in a case in which emboli starting from the neighborhood of the inflamed appendix had produced embolic abscess and suppurative pyephlebitis.[23]

An interesting case of peritonitis following appendicitis was described by Pepper, in which resolution and apparent recovery followed. Seven months later a fatal peritonitis was suddenly developed, which was found to be due to ulceration and perforation of the appendix. It is believed that perforation of the appendix may take place, followed by localized peritoneal inflammation, not of an intense character, lasting for weeks or months (Osler), the scene changing to acute peritoneal symptoms by the opening of the small localized abscess into the peritoneal cavity.

Complications.—Parotitis was met with in one case.[24] Erysipelas (recurring twice) around the abdominal incision occurred in a child aged three years.[25] Local phlebitis and thrombus, with phlebitis of the portal vein and its radicals and embolic inflammation in the liver was met with by Colquohon.

Diagnosis.—Dr. William Pepper lays great stress upon the necessity of examining per rectum to determine at an early period the existence of inflammatory tumor and pus. The differential diagnosis between retroperitoneal and intraperitoneal abscesses and between appendicitis and typhlitis, involves many difficulties. A sudden onset with intense local symptoms and peritonitis are generally due to perforated appendix. A slower development accompanies cases of typhlitis and of inflammation of the cellular tissue behind the cæcum. General peritonitis from perforated appendix is diagnosticated from other forms of perforative peritonitis by the fact that in the former the pain and tenderness begin in the right iliac fossa; there is a more gradual extension of the inflammation, and collapse is slower in coming on. Perforative and localized peritonitis have a sudden onset with fever, local pain in the right iliac fossa, and vomiting. A tumor forms in from two to five days. The difficulties of diagnosis are enhanced by the fact that even in high degrees of inflammation from perforation of the appendix, there is sometimes no local pain at the seat of the lesion. Pain in the genitals has been noted in two cases of perforated appendix. (Bryant;[26] Craig.[27])

Treatment.—In the case of appendicitis, ulceration and perforation, inducing sudden peritonitis, the question of operation is a serious one, for unless done very early, and before the peritonitis has become generalized, there is no chance of success. In a case of Pepper's[28] the abdomen was opened in sixty-four hours after the onset of the symptoms. A cavity was found filled with pus and the appendix was gangrenous.

In deciding upon the question of operation, it makes very little difference whether the pus is in the peritoneal cavity or is extra-peritoneal. In both cases exploratory incision and free opening with full drainage are the proper procedures.

The results of early operation have been so favorable that it is now a well established rule that as soon as the tumor forms it should be incised. The risk is then avoided of extension of the inflammation and rupture into the peritoneal cavity. In the event of general peritonitis having begun, the abdomen should be opened in the median line. A case is referred to in which a median incision was made, the abscess opened and its contents, consisting of fetid pus, was discharged into the general peritoneal cavity. In spite of this the patient recovered.[29]

Drs. Wilson and Goodlee[30] opened a circumscribed intra-peritoneal abscess in the right iliac fossa on the sixth day. The cavity was swabbed out with a solution of corrosive sublimate (1–500), idoform was introduced. A drainage-tube inserted, and the wound dressed with salicylic acid; although vomiting and diarrhœa supervened, forcing the intestine through the opening, the patient recovered. Removal of the perforated appendix by ligature and section was performed by J. D. Bryant[31] and R. J. Hall.[32] Peritonitis existed in both cases and both operations were unsuccessful.

Lummis,[33] in debate, referred to a case of amputation of the inflamed appendix distended with fruit seeds. Death occurred two hours after the operation. Cleveland reports a case in a child of three years. The abscess was opened about the fourteenth day and recovery resulted.

The operation is fully described in the department of *Surgery of the Abdomen.*

CONSTIPATION.

If full reference were made to all the articles upon constipation in the medical periodicals of the past year, there would be found to be much repetition, both in the methods of treating the subject and in the remedies suggested. In fact, the subject is too large a one to be treated of briefly, and experience and success with this or that cathartic combination is not all that is demanded. Constipation is a symptom of very many pathological states and its treatment is as complex as its causation; and for many reasons it is irrational to speak of it as a separate disease. The diseased conditions which have constipation as a symptom are: (1) Deficient glandular secretions (liver, pancreas, intestinal glands) due to a catarrh of the mucous lining of the intestine and bile ducts, disease of the liver and pancreas; (2) slow and not sufficiently energetic intestinal peristalsis (atony of muscular wall) due to diseases of the nervous system, central or peripheral, or to fatty degeneration of the muscular coat of the bowel; (3) congenital or acquired narrowing, dilatation or displacement of the bowel, usually of the colon. This does not include intestinal obstruction or occlusion, the result of modifications of structure in the intestinal wall, to external pressure, or fæcal accumulation within the bowel.

Before subjecting the patient to treatment the diagnosis must be definitely made.

Massage is indicated in cases in which the muscular wall of the bowel is weakened, in chronic catarrh of the intestine due to heart and lung affections, in atony of the wall of the intestine from general muscular debility, inertia, etc. Berne[106] advises that in practicing massage, pressure should be made on the gall-bladder in order to favor and excite the expulsion of bile into the duodenum. Electricity is indicated in the constipation of general nervous disease, as neurasthenia, in chronic diseases of the cord and spine, and in cases where the stools are hard and dry after cathartics are given. Electricity excites intestinal secretion. Electricity and massage combined is advocated by Köllner.[107] He prefers the weak current as being less likely to paralyze nerves. In cases of a high grade of irritability of the whole nervous system, electricity cannot be employed in the desired form or strength. Even a slight massage of the abdomen cannot be borne. Winternitz's apparatus is applicable here. A cool or cold cone (Kühlzapfen) provided with an ingress and egress tube for water is introduced into the rectum. Through the ingress tube a conducting wire is introduced into the cone; this is connected with the induced current of a battery. The other electrode, a flat plate or massage roller, is applied to the abdomen. Water is allowed to flow through the cone and is kept at a desired temperature. Leubuscher[108] has studied the effects of the galvanic and faradic currents by introducing one (the positive) electrode into the rectum, the other (the negative) being on the abdomen. The application lasted from ten to fifteen minutes. With the galvanic current an evacuation of the bowels followed in one and a half hours; after the use of the faradic current, in two and one half to three hours. The strength of the current was never great enough to induce pain, and the application lasted from ten to fifteen minutes. Fifteen cases of obstinate constipation were treated in this way. The causes of the constipation were different in the different cases, but in all there had been no movement without a strong cathartic. There were three groups of results: (1) In two patients no result [one a case of sexual neurasthenia; one of marked hysteria]; (2) in the larger number the stools became regular after treatment, but the constipation returned in from one to a few weeks after the cessation of electrical treatment; (3) in four patients the constipation was cured permanently.

Action of Cathartics.—Hess,[109] of Munich, describes his experiments to explain intestinal peristalsis and the action of laxatives. A gastric fistula was made, the opening being near the pylorus. A rubber balloon was passed through the opening and after receiving a registered amount of water was allowed to pass through the pyloric orifice and to be propelled downward by peristaltic action. The rate of progress was measured by a piece of long rubber tubing, graduated in centimetres, attached to the balloon. To ascertain the mode (or rate) of normal peristalsis, the balloon received 45 cubic centimetres of water, and a laxative mixed with ferrocyanide of potassium was administered. In 29 hours 50 minutes the balloon had proceeded a distance of 149 cm., and was stopped, it was thought, at the ileo-cæcal valve. In a second experiment 22 c.c. of water were introduced into the balloon. The passage from pylorus to anus, or 220 cm., was made in 23 hours 25 minutes. Peristalsis was intermittent, in pauses of even an hour's duration; it was more vigorous near the pylorus, and less active as the distance from the stomach increased. The less distended balloon passed more rapidly than the larger one. In the small bowels it moved more rapidly than in the colon; the relative activity during day and night was as 3:1. Expiration favored peristalsis. To illustrate the strength of the muscular contraction of the bowel, a weight of seven ounces was sustained by it when attached to the tubing outside. This force diminished as the balloon descended. To study the action of laxatives, the balloon was allowed to go some distance into the intestine and then stopped and made to act as a plug. A cathartic was now given and as long as the balloon prevented its entrance into the large intestine no catharsis followed, but as soon as by collapse of the balloon the cathartic entered the large intestine, diarrhœa resulted.

Cash[110] studied peristaltic movement in an isolated portion of intestine by introducing solid bodies and recording their rate of progress on a blackened surface by a writing lever. When the bowel was empty there were periods of complete quiescence; after food, peristalsis began and continued, reaching a minimum of activity in 4 to 5 hours. Peristalsis was excited by swallowing, by exciting the flow of saliva, and by mental impressions and exercise.

INTESTINAL OBSTRUCTION.

Frequency.—Heusner[34] maintains that yearly out of every 100,000 individuals 5 to 10 suffer from intestinal obstruction, and there is one death from this to every 300 to 500 from other causes, while Morrow[35] believes that in Albany, N. Y., there are 6 deaths annually due to this affection.

Etiology; Pathology; Morbid Anatomy.—Histories of cases have been published within the past year which illustrate very well many of the different varieties of intestinal obstruction.

Treves[36] believes that fæcal accumulation is the most common cause of chronic intestinal occlusion, and that it is most frequently met with in adult females and in association with some disorder of the nervous system. Taylor[37] reports a case of chronic obstruction of the bowels in a boy, aged 16 years, with a fatal termination, in whom the intestine was found distended by a fæcal mass consisting largely of potato-parings; and another case, with a like result, in a boy of the same age, whose sigmoid flexure was occluded by hard scybalæ. In still another case, the same author removed from the rectum a mass of fæcal matter containing pieces of broken slate pencil, bits of stick, and fragments of rag and wool, which had given rise to almost fatal occlusion. Audry[38] records a case of fatal obstruction of the bowels in a man, æt. 50, caused by the impaction in a gut of a large biliary calculus which was formed by the agglutination of several smaller ones, and he mentions that numerous cases of a similar nature are reported by Murchison, as well as fifty by Leichtenstern.

Treves considers that stricture of the colon, next to fæcal accumulation, is the most frequent cause of chronic intestinal obstruction. Knox[39] reports a case of obstruction of the bowel in a female 49 years of age, due to constriction at the splenic flexure of the colon. Acute inflammation of the colon supervened with perforation at several points and a fatal peritonitis. The author thinks that a congenital malformation may have been the original cause of the obstruction. Such strictures are frequently produced by the cicatrization of intestinal ulcers. Thus Maylard[40] narrates the history of a case in which several strictures of the intestine were found, the principal one being situated at the splenic flexure of the colon. These had been formed by the cicatrization of ulcers, many of which were found in the gut. An abscess was discovered in the abdominal parietes and the patient died of peritonitis. It was found after death that the abscess communicated with the dilated portion of the transverse colon just in front of the stricture. In Knox's case there were several plum stones in the intestine, and he thinks that the disease was rendered acute by the lodgment of one of these in the narrowed portion of the intestine. This accident was clearly to be demonstrated in a case described by Puzey.[41] In a woman, æt. 32, a non-malignant (probably syphilitic) stricture situated just below the splenic flexure of the colon had been made impermeable by the impaction in its upper part of a cherry stone.

Cancer and other new formations frequently lead to stricture of the intestine, and Treves believes that cancer, most commonly of the sigmoid flexure, is the cause of 60 per cent. of all cases of chronic obstruction of the large intestine. Schlegtendal[42] reports a case of obstruction of the bowel in a woman, æt. 30, caused by an adeno-carcinoma. Buchwald and Janicke[43] had under observation a unique case of intestinal occlusion in a boy of 6 years, which required laparotomy. Two entero-cysts (weighing together 875 grams) were found. Their weight produced complete stenosis of the jejunum, to which they were intimately connected.

Strangulation of the intestine by bands, the result of peritoneal inflammation, is a morbid condition of which examples have been recently reported by Heusner[34] (in a case of tubercular peritonitis), Morrow,[35] Cilley,[44] Clark,[45] Harris,[46] and Waitz.[47] In all the small intestine was the part involved, and in the case described by the last mentioned author, the obstructing band was in the sac of an umbilical hernia. Less frequently the vermiform appendix has produced strangulation, as in the cases of Harris,[46] Davidson,[47] and Rehn.[48] In all of these there was peritonitis, which in the first two was due to perforation of the intestine and extravasation of fæces. Rehn's case concerned a seven months' gravida.

A portion of the mesentery may in the same way produce occlusion, as in the case mentioned by Pearce,[49] where the ileum in a boy, 9 years of age, was strangulated for six inches of its course.

Interesting instances of internal incarceration have likewise been described, in which the gut has passed through some normal or abnormal opening. Thus in St. Thomas' Hospital Reports a case is cited in which a knuckle of colon and a mass of omentum had passed through an opening in the diaphragm and become strangulated; a case by Cattiaux[50] where a loop of the small intestine had penetrated through a hole in the mesocæcum; and one by Rehn,[48] of incarceration in the obturator canal. Heusner[34] describes an interesting case in which one portion of the bowel was strangulated by another. He observes that when obstruction is due to knotting of two intestinal loops, symptoms of occlusion appear early, and a fatal termination may be expected in a very short time. Pressure from enlargements external to the intestine may likewise induce occlusion. Morrow[35] narrates the history of a case where this was brought about by an enlarged mesenteric gland covered with lymph, and Aly[51] an instance of intestinal obstruction produced by the pressure of an abscess originating in a suppurating peritoneal gland. Invagination causes, according to Heusner,[34] one-third, and in children three-fourths, of all intestinal obstructions, while Treves[36] considers that chronic intussusception is a less frequent cause of this affection than fæcal accumulation, stenosis of the colon from stricture, or stenosis of the small intestine from causes other than stricture. Yet invagination has been the occasion of a large number of cases of intestinal obstruction recorded within the past year. Of the histories of cases of intussusception submitted to us, one-fourth part concerned children under one year of age, while once a female aged 74, and once a male aged 86 years, was the subject of this disease. Tumors of the intestine seem to be a not infrequent cause of intussusception. Lane,[52] in a child of 8 years, found an invagination in which the transverse colon was the intussuscipiens. A tumor originating in the mucous membrane of this part of the bowel projected from the rectum. Barker,[53] Bryant,[54] and Kulenkampf,[55] likewise met instances in which a tumor (in one case an adenoma or epithelioma, and in another a papillomatous and probably malignant growth), seated at the apex of the sigmoid flexure, produced an invagination into the rectum. Here again it was possible to draw the tumor out of the anus. The same pathological condition, involving, however, all of the large intestine and part of the ileum, followed a linear rectotomy, performed by Guillet[56] for the removal of small, pediculated, adenomata scattered throughout the large intestine. The intussusception was irreducible, and the patient died.

Other causes, however, may be of force in the production of intussusception, and Hagar[57] met with an instance in which he believed that the ascending colon (the part invaginated) had been injured in a fall which the individual had sustained one year before the occurrence of the attack, and that the injury had led to a permanent change in the nutrition of the intestine, ending in paresis. Tateo, likewise, encountered a case in which an invagination was due to weakness of intestinal innervation, but there was no history of previous injury.

That intussusception may be cured spontaneously by sloughing and subsequent discharge of the obstructing part is a fact well illustrated by a case reported by Sticker.[58] Urgent symptoms of ileo-colic intussusception appeared but were dissipated by intestinal irrigation. Six months later the individual died, and it was found that there had been an invagination, and that part of the slough had been discharged, leaving an opening at the neck of the intussusception between the ileum and cæcum. Fæcal impaction and intestinal perforation above the old invagination was discovered to have been the cause of death. Lauenstein[59] found on autopsy in a case of intestinal obstruction, a volvulus which could be untwisted, and which had apparently been relieved during life by intestinal puncture and rectal injections.

Diagnosis.—Kümmel[60] considers that the diagnosis is the most difficult feature connected with the whole subject of ileus, and he calls attention, as do also Davies Colley[61] and Stewart,[62] to the close resemblance between the symptoms of intestinal obstruction and those of peritonitis. Heusner[34] likewise observes that perforative peritonitis, by paralyzing peristalsis, gives rise to disturbances simulating those which attend occlusion of the bowel and may even induce fæcal vomiting. Indeed, he gives the histories of two cases with symptoms of intestinal obstruction in which laparotomy was performed; in both perforative peritonitis but no occlusion was found. To aid us in distinguishing between these two conditions, Treves[36] reminds us that in peritonitis we have ascites, continuous pain, a significant posture of the patient, tenderness of the abdomen, constant and "meaningless" vomiting, early distension of the abdomen, and complete absence of visible intestinal coils. When symptoms of intestinal obstruction become manifest the cause can, according to Déprès,[63] be determined in 99 out of 100 given cases. The following diagnostic features of the different forms of chronic intestinal obstruction are emphasized by Treves:[36]

(1) Fæcal accumulation is most frequently met with in adults and especially females, and is often associated with a disordered nervous system. It begins as an increasing constipation which may become marked before symptoms of colic appear. There is loss of appetite, a foul tongue, disagreeable breath, nausea, flatulence, paroxysmal pain and fæcal tumor. Should vomiting occur, it is late, scanty, and not fæculent, unless obstruction has continued for a considerable length of time. (2) In stenosis of the colon the symptoms and course are much the same as in stenosis of the small intestine. There is intermittent colic and constipation, though diarrhœa with tenesmus may be present and meteorism is marked. (3) The previous history is of importance in the diagnosis of stenosis of the small intestine. The onset is gradual, and acute or

subacute attacks of obstruction occur now and then. There is intermittent pain with nausea and vomiting if the attacks are severe, though the latter is rarely fæculent. In 40 per cent. of all cases constipation alternates with diarrhœa. The abdominal walls are relaxed and not tender, unless there be peritonitis; there is emaciation and exhaustion, but no meteorism. The sex and age of the patient are unimportant diagnostic features. (4) The essential points in the diagnosis of chronic intussusception are the youth of the patient (children and young adults being most often the subjects of this affection); the onset, which in 60 per cent. of all cases is insidious; the invagination tumor, encountered in 50 per cent. of chronic cases; blood in the stools, in 50 per cent.; intermittent colicky pain; the absence of vomiting in 50 per cent. (rarely stercoraceous vomiting); bowels irregular with a tendency to diarrhœa; and the absence of meteorism.

According to Steele,[64] more than one-half of all cases of intussusception occur within the first ten years of life, and one-quarter within the first twelve months. In infants, this affection is most frequently encountered in the fourth, fifth, sixth and seventh months of life.

Hagar[57] directs attention to the importance of many of the diagnostic signs of intussusception mentioned above by Treves. As symptomatic of acute intussusception he also refers to the sudden onset and the tenesmus and muco-sanguinolent discharges. Ollive[65] indeed, believes that in a previously healthy child, hæmorrhage from the bowels is usually significant of the presence of an invagination. Hagar, as well as Treves, speaks of the invagination tumor as a valuable element in diagnosis, but observes that although this may be discovered easily in many instances on account of the laxity of the abdominal walls, yet when absolute constipation exists, pronounced meteorism may occur early, and render our efforts to discover it abortive. Ransohoff[66] maintains that in most cases of intussusception there is the appearance of great anxiety, but that true collapse, with rapid and feeble pulse, and cold, clammy, perspiration is not present.

Treatment.—Treves[36] recommends, in chronic stenosis of the small intestine, a simple diet consisting of easily digested food of such a character that the quantity of solid matter in the intestine will be reduced to a minimum. Plenty of liquids should be taken, and cod-liver oil. Aperients are to be avoided, but massage of the abdomen is to be employed with copius enemata; during acute attacks, rest, starvation, and opium. In this affection, Havilland Hall[67] employs rectal suppositories of beef peptonoids every four hours, the rectum being washed out twice daily with tepid water. To prevent obstruction from fæcal accumulation it is important that a patient suffering from chronic constipation should receive an abundance of liquids. At the end of an acute attack, when the bowel begins to clear, he advises that 5–10 grains of calomel be prescribed and subsequently a saline. Allingham, Jr., has observed that in some cases of fæcal accumulation benefit follows forcible dilatation of the sphincter ani.

Lavage of the stomach in the treatment of ileus, a procedure first suggested by Kussmaul, has found many strong advocates. According to Schlegtendal[42] it fulfills a three-fold therapeutic indication: it prevents disagreeable symptoms, alleviates them when they have already appeared, and in some cases cures the disease. By this means the stomach is emptied of its contents and also, to a certain extent, the intestine above the point of constriction, as Rehn[48] observed in two cases where the effect of washing out the stomach was watched, the peritoneal cavity being opened. Heusner says that by this means many litres of intestinal contents can be removed. Pain is alleviated, eructations and vomiting are controlled, violent peristalsis is quieted, the function of the stomach is restored, suitable nourishment can be taken and assimilated, thus maintaining strength and life until the lumen of the intestine is again brought to the normal degree of patency. He has also observed a diminution of meteorism and a favorable influence in collapse.

To the four methods by which washing out the stomach produces good results in ileus, as formulated by Cahn and Hasenclever[69]—emptying of the stomach, lessening of intestinal distension, quieting of peristalsis, and stimulation of the splanchnic—Schlegtendal adds a fifth. He believes that washing out the stomach (and duodenum) is of service in aiding nature, striving by anti-peristalsis to get rid of the fæcal accumulation above the point of obstruction. Thus the abnormal pressure above this point is rapidly reduced, and the intestines are enabled in favorable cases, by an independent reposition movement, to regain their normal position and relations.

Two conditions which cannot be relieved by irrigation of the stomach, although symptoms attending them may be mitigated in severity, are, as pointed out by Heusner, obstruction produced by the compression of new growths, and strangulation in internal hernial and peritoneal pouches. Lenhartz[70] says that this mode of treatment should be resorted to in cases of acute intestinal obstruction in which injection of the bowel has failed, before resorting to operative measures, and Treves recommends it in cases of stenosis of the small intestines. Bardeleben, Schlegtendal, Heusner, Lenhartz and others refer to the other, the unfavorable side of this question, believing that washing out the stomach, by relieving urgent subjective symptoms, induces the surgeon to postpone operative interference until valuable time has been lost, gangrene of the affected portion of the gut has taken place, and the proper time for surgical intervention has passed by. The same objection, however, as the last observes, obtains with the opium treatment of ileus. Still, washing out the stomach in obstructions which are very acute and entirely unexpected, even when there follows no improvement in the morbid condition present, is, according to Schlegtendal, of great service in preparing the patient for subsequent operation. Madelung, as Rehn asserts, is of the opinion that progress in laparotomy for ileus can only be made when some means is discovered of previously emptying the overfilled intestine of a part or the whole of its contents before the search for the obstruction is undertaken. Surely washing out the stomach achieves this result most admirably. Further than this, the procedure under discussion is sometimes of material assistance to the operator after the peritoneal cavity has been opened, as was shown in a case reported by Rehn. Here the obstruction was due to a portion of the intestine, which could not be brought to view, being fastened in the obturator canal, and the coils of distended intestine protruding from the abdominal wound further embarrassed the operator, and could not be reintroduced into the abdominal cavity. After washing out the stomach, the intestines were emptied and could be replaced, and the incarcerated portion of the intestine could easily be lifted up.

The following cases will illustrate practically the principles which have just been referred to:—

Schlegtendal[42] reports a case of obstruction of the intestine due to carcinoma, in which washing out the stomach was followed by excellent results. Fifteen hours later the symptoms reappeared, but a repetition of this procedure failed to relieve them. Wentscher,[71] a case of occlusion lasting ten days, which was cured by one washing out of the stomach preceded by intestinal puncture. Kussmaul[72] kept a patient alive 23 days and until the obstruction in the intestine was relieved. Stewart[62] reports a case of intestinal obstruction in which after washing out the stomach there was no vomiting for 12–15 hours, although it had been stercoraceous previously. Lando[73] reports the case of a man, who for ten days had obstipation, hiccough, fæcal vomiting, etc. The stomach was washed out twice daily for 10 days. After each washing the hiccough and vomiting were relieved for several hours. The bowels were opened on the tenth day and the patient recovered. There was supposed temporary paralysis of the muscular coat of the bowel. Jacos[74] records a case of continuous vomiting and obstipation due to supposed occlusion of the gut. The stomach was washed out with iced water. This procedure was followed by violent pain in the stomach, the passage of flatus, and the cessation of vomiting. It was once repeated. The bowels were opened and the patient recovered. Rectal injections had proved inefficacious.

Herz[75] believes that many cases of intestinal invagination in children can be reduced if relaxation be secured by anæsthetics and gentle abdominal taxis be employed. If these fail, he recommends the injection of a large volume of air or water. The child should be laid upon its back, and through a rubber catheter introduced as far as possible into the intestine, air should be forced for two minutes, while gentle massage at the supposed seat of the invagination is performed. Steele[64] recommends that the intestine be inflated with as much air as it will stand, thrown in by a double bulb syringe, while Ollive[65] considers that this procedure should be resorted to as soon as intestinal hæmorrhage occurs in a previously healthy child. Steele further suggests that in intussusception in infants opium be given, the child be chloroformed, and a rectal tube be introduced for a distance of six inches within the anus. This is connected by rubber tubing with a glass funnel into which are alternately poured solutions of bicarbonate of soda and tartaric acid (of each three drachms). The carbonic acid gas evolved distends the intestine. Tateo[76] maintains that the treatment of intestinal occlusion by the injection of large quantities of water originated with Lorenzo Marsoni in 1761, and was later perfected and generally recommended by Catani. Herz suggests that if water is to be injected, the child should first be given a warm bath of one hour's duration, should then be anæsthetized, and three or four litres slowly forced into the bowel. If this fails, one litre of very cold water may be substituted. Steele believes that injections of warm water should be resorted to (to be given with as much pressure as the bowel will bear), if inflation with air and carbonic acid gas have failed. Treves recommends copious enemata in obstruction due to stenosis of the small intestine, and the same, in the knee-elbow position with massage of the abdomen, when due to fæcal accumulation or stenosis of the colon. Lenhartz, says truly, that high injections in the treatment of intestinal obstructions are generally dispensed with after two or three attempts have been followed by no result, and Steele insists that the measures above recommended by him, with inversion of the child, abdominal massage, and taxis, should be persevered in for from 24 to 48 hours, according to the age and condition of the child. In intussusception in children, injections of warm soapsuds, according to Mueller, are better than air, for the former aid the patient in rallying from shock and nervous depression, and distend the gut below the obstruction, while the soap lubricates the parts, is soothing to the inflamed tissue, and softens hardened fæces, should any be present. Tateo believes that in cases of obstruction due to strangulated herniæ, one cannot expect a cure from intestinal injections. Still, they are worth a trial before proceeding to operative measures.

Instances of obstruction of the bowel where intestinal insufflation or irrigation have been employed with more or less success have been reported by Hemenway,[77] Herz,[75] Lenhartz,[69] Sticker,[58] Stewart,[52] Morrow,[35] Munro,[78] Sée,[79] Jacos,[74] Elliot,[80] Cheadle,[81] Hagar,[57] Tateo,[76] Waitz,[82] Lauenstein,[83] and others.

Lenhartz[43] advises that in cases of ileus not yielding to other means, the distended gut should be repeatedly punctured, before other more serious operative measures are instituted, and Wentscher[44] reports one, and Curschmann[84] three cases of cure following this procedure.

Kümmel[85] believes in intestinal puncture from a theoretical stand-point, for he says that if the greatly distended gut is unburdened of gas and pressure is relieved, it becomes relaxed or collapsed, and it is thus possible for its permeability to be reestablished. Practically he has never had the courage to resort to this measure. He considers it extremely difficult to find the loop sought after and to puncture it, and the puncture of an undesirable portion of gut is liable to be followed by an extravasation of fæces. This danger is referred to by Lenhartz, but he did not meet with this accident in either of two cases in which he had recourse to the measure. Aly, however, reports the history of a case in which life was threatened from the pressure of great meteorism. A distended intestinal loop was punctured and a large quantity of gas escaped. The patient died, and on autopsy it was found that there had been some fæcal extravasation from the punctured opening. Hess[86] says that since age exercises an influence upon the tonus of the intestine, puncture should only be resorted to in strong middle age.

The following is an outline of the views entertained by different authors in regard to the graver operations performed for the relief of intestinal obstruction:—

Heusner:[34] In cases of occlusion of the bowel which run a relatively chronic course, and which are due to new growths or the results of peritoneal inflammation, operation gives fairly good results. One should not resort to operation hastily, for many apparently critical cases recover without it. Braun:[87] Of 63 cases in which laparotomy was performed for intussusception, only those 12 recovered where disinvagination could be brought about; but where resection of the gut was done, or an intestinal fistula was made, the termination was fatal. Lenhartz:[43] Even when an internal strangulation is diagnosticated early, if the cause of the obstruction is uncertain, laparotomy should be resorted to only when other means fail and life is threatened. Steele:[64] In intussusception in children, if no relief follows simpler measures, laparotomy should be performed (in children under three years of age), and if the gut is gangrenous, it should be stitched in the wound and an artificial anus made. This procedure gives better chances for life than primary enterectomy. Stewart:[62] Operation in the first 24–72 hours is the only hope in cases of intussusception. Treves:[36] In cases of stenosis of the small intestine, after the failure of other means, the patient getting steadily worse, laparotomy should be performed and adhesions freed, the gut resected, or an artificial anus made. In cases of stenosis of the colon, if enemata and aperients fail, operative measures should be undertaken: lumbar colotomy is the best; colectomy if an actual stricture is discovered. In all cases of intestinal obstruction where there is doubt as to the nature or seat of the occlusion, an exploratory abdominal opening should be made in the median line. Richelot:[88] If the cause of obstruction is known, make an artificial anus; if not known, perform laparotomy and search for it. In cases where no diagnosis is possible, Verneuil immediately makes an artificial anus, while Trélat and Le Fort prefer laparotomy. Czerny:[89] If diagnosis is difficult, make an artificial anus; this operation is to be unconditionally preferred to the ordinary operation of laparotomy.

The following is a summary of the cases in which one of the graver operations, just mentioned, was performed:—

Heusner:[34] Laparotomy in a case in which one portion of the bowel was strangulated by another. The strangulation was relieved, and 5 minutes later there was a passage of fæces. Death from collapse in the first 24 hours (8th day of the disease). The same: Laparotomy; ileum strangulated by a band; tubercular peritonitis; recovery; death two years later from phthisis. Stewart:[62] Laparotomy on the 9th day of disease for intestinal obstruction due to glueing together of two intestinal loops; diffuse peritonitis was found with local accumulation of pus in hypogastric region; obstruction freed; drainage-tube; death five hours after operation from shock. Buchwald and Jannicke:[43] Complete obstruction caused by enterocysts; laparotomy; affected portion of the bowel excised; death from heart failure. Morrow:[35] Obstruction in a child; lasted one week and defied other treatment; laparotomy; pressure of a gland and a band relieved; death in four hours. Czerny:[89] A boy with an internal incarceration; intestine opened and artificial anus made; recovery. Knaggs:[90] Intussusception in a young child; resection of the gut; death in a few hours. Teale:[91] Intussusception in a man, æt. 86; resection of gangrenous portion of gut; death from exhaustion on sixth day. Hagar:[57] Intussusception in a boy; laparotomy; bowel torn in attempting reduction; resection of affected part; ends united;

death from shock three-quarters of an hour after operation. Rehn:[48] Strangulation in obturator canal; exsection of gangrenous portion of intestine; death in collapse on following day. The same: Laparotomy in a woman four hours post-partum; strangulation by Merckel's diverticulum; diffuse peritonitis found; death. Waitz:[46] Obstruction by a band in a large umbilical hernia; sac opened; band divided; recovery. It will be seen that the mortality from these more grave operations for the relief of intestinal obstruction is very large.

PERITONITIS.

Etiology; Pathology; Morbid Anatomy.—Foxwell[111] pleads for the recognition of acute primary peritonitis as a distinct affection. He believes that it is a disease of low mortality, and when death occurs, the peritonitis is very prone to be considered as secondary to coexisting lesions. According to the author, in a large number of instances, peritonitis, along with pneumonia, pleurisy, and pericarditis, may be regarded as the semi-independent effect of a common cause. This cause may be—excluding traumatism—general septic infection, Bright's disease, a specific fever, and especially exposure to cold and wet. A very apt illustration of this view is furnished by the history of a case reported by Robinson.[112] Here a general peritonitis occurred in the course of an attack of acute articular rheumatism, along with double pleurisy and pericarditis. Further support is to be found in a communication by Froelich,[113] in which he reports the case of a soldier who was attacked with lacunar tonsilitis. Subsequently peritonitis developed and the patient died. Autopsy showed adhesive and purulent peritonitis, which the author believes was caused by the cold to which the tonsilitis owed its origin. He considers that both of these conditions were brought about by the same micro-organisms. In support of this opinion he asserts that during the autopsy he received a wound of the finger, and after swelling of the injured member and of the axillary glands, well-marked symptoms of lacunar tonsilitis appeared. His wife, who had been confined to the house for some time previously, and therefore had not been exposed to the weather, developed the same disease, as did also a helper at the autopsy who had punctured his thumb with a needle.

Regarding *tubercular disease* of the peritoneum, Deschamps,[114] in a recent clinical lecture observed that the peritoneum could be infected with tubercular matter either directly, as by wounds, or indirectly. Direct infection, which he believes to be uncommon, could occur (1) through the intestinal canal,—in which case the bacilli enter with the food; (2) through the blood, when tubercular disease exists elsewhere; (3) through the pleuropulmonary or diaphragmatic channels, when the lung is primarily affected, then the pleura, the bacilli thence reaching the peritoneum through the lymphatics of the diaphragm; (4) through the genital organs, *e.g.*, the ovary; (5) from the iliac glands, as in cases of tuberculosis of the hip.

A number of cases of *perforative peritonitis* have recently been reported. Thus, Rothmann[115] mentions an instance of fatal peritonitis caused by perforation of a round ulcer of the duodenum in a patient with membranous enteritis, and Ord,[116] a case where the gall-bladder had been opened by an ulcer on its upper surface. Stewart[117] met with a case having a similar termination, in which a perforating ulcer on the anterior wall of the stomach near the pylorus produced peritonitis. Mesnard[118] records a case where rupture of the stomach followed by fatal peritonitis occurred in a man with cancer of that organ. He believes this accident to be rare, and can find but two other cases published.

The effect of various agents in the causation of peritonitis has recently been independently investigated by Grawitz[119] and Pernice.[120] The conclusions arrived at by the first of these may be briefly summarized as follow: Schizomycetes or the staphylococcus aureus injected into the peritoneal cavity will not of themselves produce peritonitis, if the injecting medium be non-irritating. In the majority of cases, two things are necessary for the causation of suppurative peritonitis: (1) the introduction of pus-producing organisms, and (2) the existence of some local disturbance, as when the intestinal tube is gorged with stagnant matter, or the serous membrane has been denuded of its epithelium. In sharp contrast to the opinion of Foxwell, previously given, is the view entertained by Grawitz, that cold alone cannot lead to peritonitis, nor can it be regarded as an important local factor when other possible causes are present.

Pernice endeavored to discover by experiment whether chemical agents, without the coincident action of micro-organisms, could cause peritonitis. He found that injection into the abdominal cavity of small quantities of the mineral acids, acetic acid, phenol, concentrated sublimate solutions, and nitrate of silver, produced the disease, but the exudate was always serous or sero-fibrinous, and never purulent. Finally, Chevalier[121] maintains that peritonitis may be produced by blows inflieed upon the abdominal wall in cases where no local evidence of the injury is apparent during life, or when the disease is fatal, after death. He narrates the histories of three cases, one of which ended in recovery, in support of this opinion.

Symptomatology and Diagnosis.—Goodhart[122] gives the histories of several interesting cases of peritonitis, and calls attention to the uncertainty of some of the so-called diagnostic features of this disease. He believes that in acute plastic and suppurative peritonitis the temperature may give no sign, although in a majority of cases it is an important factor. Still, the temperature of peritonitis is uncertain, and in a large number of instances it shows no elevation. This opinion is corroborated by the experience of Chevalier, who found in three cases of peritonitis, caused by blows received upon the abdomen, no elevation of temperature, although the pulse was rapid, small, and compressible, and pathognomonic signs of peritonitis existed. He regards the absence of fever as due to the persistence of the nervous shock consecutive to the blow. Dreschfeld,[123] too, in peritonitis due to perforation of the stomach, even when the inflammation is extensive and there is exudation of lymph, believes that the temperature remains normal or only rises slightly, to fall again below normal when collapse supervenes. Another negative symptom, Goodhart thinks, is also likely to deceive, namely, the absence or comparative absence of pain. When pain is present, its location is by no means constant. Although, according to the same author, constipation is more common in this affection than diarrhœa, yet the latter does not negative the existence of peritonitis, especially the suppurative form, and when present, must be regarded as a grave, almost fatal omen.

Of tubercular peritonitis, Deschamps says that when the disease is introduced through the intestinal canal, the primary symptoms are diarrhœa, nausea, vomiting and abdominal pain. The pain is frequently located at the level of the junction of the ascending and transverse colon, a favorable situation for the arrest and development of bacilli. Here a fæcal tumor is apparent: from this point the disease invades the abdominal walls, the peritoneum or the under surface of the diaphragm, and thence the pleura. The clinical picture does not always vary with the mode of invasion. Ascites will be a prominent symptom if the bacilli develop around vessels and compress them; jaundice, if the bile ducts are pressed upon; sciatica, if the sciatic nerves are subject to pressure. (Case cited of double sciatica from this cause.) The general aspect of the patient will depend more upon the rapidity of progress of the tuberculosis than upon the mode of invasion.

Plenio[124] alludes to the importance in the diagnosis of perforative peritonitis of the presence of a layer of air between the abdominal parietes and the intestinal tract, and the consequent absence of perceptible peristaltic movement at the affected place. This sign, described by Wagner, he had a chance to verify in a case of perforative peritonitis twice operated upon. Dreschfeld regards marked change in respiration as the first symptom of peritonitis incident to perforation of the stomach. The pulse then increases in frequency, and becomes small, thread-like, and running. There is cold, clammy perspiration, pinched features, a diminution in abdominal pain, and collapse, while consciousness is preserved until the last. The effect of peritonitis in paralyzing peristalsis, and therefore leading us to believe that the case is one not of peritonitis, but of intestinal obstruction, has been referred to in treating of this latter affection, and is also commented upon by Goodhart and Thoman.[125] The last named author narrates an interesting case of acute, primary, miliary tuberculosis of the parietal and visceral layers of the peritoneum, simulating an acute intestinal obstruction.

That adherent intestines from peritonitis may resemble a fibroid tumor of the uterus is proven by a case which Price[126] relates.

Prognosis.—Deschamps believes that one is prone to make too gloomy a prognosis in cases of tubercular peritonitis, which in certain instances, particularly if invasion has taken place through the genital or intestinal canals, he considers curable. Most cases of peritonitis brought about by perforation of the stomach, according to Dreschfeld, end fatally. Occasionally recovery takes place where the opening in the stomach is small and valve-like.

Treatment.—Tait[127] advocates the use of purgatives in that form of peritonitis which follows surgical operations. But he believes that to be of any use they must be employed early, before the intestines have been paralyzed by abdominal distension, and the stomach exhausted by excessive vomiting. Treatment by purgatives, he says, is intended to abort rather than cure peritonitis, but advanced peritonitis may be cured if vomiting can be controlled. This may be brought about by giving the stomach absolute rest for 24 or 48 hours, while morphia is administered hypodermically. Wilkinson[128] thinks that the use of blisters is a mode of treatment in peritonitis which has not received the attention from the profession commensurate with its merits. In tubercular peritonitis Deschamps also recommends repeated vesication and revulsives. Lavage of the stomach, which has been found so useful in the treatment of intestinal obstruction, has likewise been employed by Lewin[129] in two cases of acute peritonitis, with the most excellent results. The stomach was washed out thrice daily and opium given afterward. Immediate relief was obtained, pain and eructations were lessened, sleep procured, and meteorism diminished. Goodhart remarks that the treatment in peritonitis is generally the same,—opium, belladonna, and a restricted diet; but that some cases which seem critical, recover, and others which appear not so grave, die. He clearly pictures the dilemma in which every physician finds himself now and then, when he is unable to decide whether to trust alone to medicinal treatment or to run the risk of an exploratory incision. When cases die and no operation has been performed, he asks himself, "Could life have been saved by opening the abdomen?" The deaths make him think, "I wish I had done something;" the recoveries, "I am glad I waited." The author believes, however, that if symptoms are severe and the disease seems to be localized, an operation should be performed. Truc[130] on the whole agrees with this advice, regarding operation justifiable when the patient's life is threatened; but he considers collapse and hyperpyrexia as contra-indications to formal surgical interference. Trzbickey,[131] from experiments made upon dogs, concludes that operative treatment is indicated in cases of purulent or septic peritonitis, and that salicylic acid is by far the best antiseptic for use in irrigating the abdominal cavity. Treves[132] thinks that in acute general peritonitis abdominal section should be made as soon as the diagnosis is established, and he characterizes temporizing as futile, purposeless, and reckless. But he considers this operation inapplicable in peritonitis in connection with carcinoma, general septicæmia, or extensive rupture of certain of the viscera. Dreschfeld[133] and others discuss the propriety of laparotomy in cases of perforate peritonitis. Treves,[134] Taylor,[134] Roberts,[135] Barwell,[136] Taylor,[137] record cases which show the beneficial effect of laparotomy in different forms of peritonitis.

The applicability of abdominal section in the tubercular form of peritonitis is discussed editorially in the *Medical Press and Circular*, and the opinion is formulated that, as a rule, in this affection it is best to open the abdomen and cleanse the peritoneum, and that when the presence of pus is established, due to this or any similar cause, the cavity of the peritoneum should be treated as would any other serous cavity under like conditions. In this Knaggs[138] also agrees, regarding the outlook in those cases especially favorable where there is no visceral involvement.

Both Van de Warker[139] and Truc[140] maintain that laparotomy in tubercular peritonitis cannot be considered a dangerous operation; but according to the latter, it will not prove efficacious in the diffuse form of the disease, but is more useful in the localized chronic form with effusion. Truc believes that this procedure is especially indicated when the general condition of the patient is suffering; when there is fever, loss of appetite and exhaustion; when respiration is embarrassed by mechanical pressure; and when there is danger of an opening externally or into one of the hollow viscera. Marked exhaustion and visceral lesions of excessive gravity, he regards as the only contra-indications to laparotomy. Treves observes that the operation has never been successful in young children. Many have sought to discover why it is that opening and cleansing the peritoneal cavity retards or arrests progressive tuberculization of this serous membrane, but so far no satisfactory conclusions have been reached. That it does have this salutary effect is unquestioned. Abundant evidence is furnished in the cases reported severally by Van de Warker, Knaggs, Clarke, Homans, Truc, and others. The instances cited at a recent meeting of the Congress of German Surgeons would alone be convincing. But further than this, the beneficial effect upon the general condition of the patient and upon concomitant pulmonary lesions of exposure and irrigation of the peritoneal cavity is sometimes startling, and has been commented upon by nearly all the observers whose names have been already quoted. While most operators regard the use of the drainage-tube after laparotomy for the various forms of peritonitis as expedient or essential, Barwell cannot well see how the lower part of the peritoneal cavity can be drained through a tube having its outlet on the anterior surface of the abdomen. He advises that no drainage-tube be inserted, immediately after the operation, but if distension occurs subsequently, the lower stitches may be removed and an outlet given to the fluid accumulation. The tendency of serous and purulent accumulations within the peritoneal cavity to find a vent through the abdominal wall has been observed by Hochhaus,[141] Augener,[142] Shattuck,[142] and by others.

Ascites.—Straus[101] reports an interesting case of chylous ascites in a man aged 61 years. The abdominal cavity was tapped three times and fluid withdrawn. The fluid evacuated at the first and second tapping closely resembled chyle; that at the last tapping contained a larger proportion of fatty granules. It was proven that this increase was due to the larger amount of fat ingested by the patient. On post-mortem examination it was found that the omentum, parietal peritoneum, and other of the abdominal contents were cancerous. There was extravasation of chyle beneath the serous coat of the small intestine. The author deduces the following points in regard to the pathology of the disease. In this case there was impermeability of the mesenteric glands and obstruction of the lymphatic vessels by cancer. There resulted: (1) stasis of chyle in the villi and mucous membrane of the intestine; (2) extravasation of chyle beneath the serous coat of the small intestine and probably into the peritoneal cavity; (3) rupture of lymphatic vessels in the mesentery forming chylous or lymphatic fistulæ. The author, in conclusion, believes that there exists a distinct variety of ascites due to effusion of chyle into the peritoneal cavity, which always results from an obstacle situated in the course of the lymphatic vessels, lymphatic glands, or the thoracic duct. The chyle finds its way into the peritoneal cavity by the rupture of lymphatic vessels or transudation through them. In support of this view a case recorded by Secretan[102] may be mentioned. In this case a woman, æt. 58, with chylous ascites, had tubercular deposits in the mesentery and peritoneum, in the genitalia, liver and intestine.

The further recent literature on the subject of ascites may be briefly summarized as follows:—

Antona[103] recommends continuous drainage in ascites and gives a case where this proved of benefit. Micelli[104] records a case of ascites *à frigore* in a young woman where paracentesis was performed 34 times. After the last tapping there was no further reaccumulation and the patient subsequently enjoyed good health. Grancher[105] mentions a case of ascites in a child aged 6 years caused by cirrhosis of the liver.

DISEASES OF THE MESENTERY.

Van Bibber[143] reports three cases of cancer of the mesentery, in one of which the disease was supposed to be abscess of the spleen; in another, stricture of the rectum; and in the third, cancer of the omentum. This affection forms 14.7 per cent. of all malignant abdominal diseases. One important diagnostic symptom is deep-seated, constant pain, sometimes acute and lancinating, at other times dull and wearing in character, and always referred to the region of the mesentery.

Koefoed[144] reports a case of lipoma in the mesentery of the small intestine in which an explorative laparotomy was performed, but the tumor could not be removed. Vergely[145] cites a case in which a diagnosis of cancer of the mesentery was confirmed upon autopsy.

Hahn,[146] in a comprehensive paper upon mesenteric cysts divides these new growths into (1) blood cysts, (2) chyle cysts, (3) serous cysts, and (4) hydatid cysts. He reports an interesting case which occurred in a girl æt. 8 years. Abdominal section was performed, the cysts evacuated, and the cyst wall dissected away. The wound in the mesentery was united with catgut. The child recovered. In the discussion which followed the reading of this paper Küster and Gottschalk alluded to cases of cysts of the mesentery which had come under their observation.

Bramann[147] reports a case of chylous cyst as large as a child's head, found on laparotomy in the mesentery of the small intestine. The cyst was evacuated and its edges were sewed to the abdominal wall.

GASTRO-INTESTINAL AND INTER-INTESTINAL FISTULÆ.

Ulcer and cancer of the stomach occasionally lead to the formation of a fistulous connection between the stomach and different portions of the intestinal canal, with the transverse colon most frequently, more rarely with the duodenum or jejunum. Dreschfeld[99] refers to Murchison's record of 33 cases of gastro-colic fistulæ (21 of which resulted from cancer; 9 or 10 from ulcer), and reports 2 cases of his own. The diagnosis of this condition is based upon the previous history of cancer or ulcer, the cessation of the characteristic vomiting, fæcal vomiting taking its place, and the escape of half-digested food from the bowel. The stool follows soon after the ingestion of food. Schönlein suggested to administer food colored with cochineal and water in order to determine the time taken to pass from the stomach to the rectum. The prognosis is unfavorable both in cancer and ulcer. Gastro-duodenal fistula is rarer: three cases only of this are recorded. Inter-intestinal fistulæ occur from perforation of the bowel after adhesions have formed in typhoid fever or tuberculous disease. Murchison refers to three cases of this. Dreschfeld adds two of his own. In one a fistula connected the cavities of the stomach, the colon, and duodenum.

INTESTINAL ANTISEPSIS.

That the absorption of putrid matter from the intestinal canal is capable of producing symptoms of intoxication is well known. That fæcal matter, presumably healthy, is also able to induce disturbances by absorption, is not so generally admitted. Bouchard[148] believes that he has demonstrated that the extract of 45 grams of fæcal matter is sufficient to intoxicate a kilogram of animal. In a putrid state, 17 grams is sufficient to produce the same result. In intestinal indigestion and acute and chronic intestinal catarrh, the contents of the intestine undergo putrefactive decomposition with the development of toxic principles, as leucin, tyrosin, ptomaines, phenol, iodol, skatol, etc. These may be supposed to be the probable cause of grave disturbances of the nervous system which accompany the innutrition and anæmia of impaired intestinal digestion. Among the most marked and obstinate forms of nervous disturbance which accompany profound anæmia is the intense thoracic neuralgia which often simulates angina pectoris with syncope. This is usually attributed to anæmia only, but its intensity and occasional occurrence give ground for looking for some specific cause; and it may be that a toxic agent absorbed from the intestine is this cause. In such cases a remedy must be sought for which will be an intestinal antiseptic preventing putrefaction. Semmola employed the alkaline and earthy sulphates for this purpose. Charcoal, iodoform, corrosive sublimate, creasote, salicylate of soda, bismuth, carbolic acid, sulpho-carbonated water, and naphthalin have been suggested and tried. Martini employed thymol in doses of 20 to 120 grains a day in all cases—as diarrhœa and dysentery—in which fermentative action was kept up in the intestine. The proof of its good effect is seen in the disappearance from the urine of phenol, which is one of the most constant products of intestinal putrefaction in health. Bouchard advises naphthol on account of its insolubility, which increases the chance of reaching the locality of putrefactive change. 35 grains can be given in divided doses during the day, in emulsion, pills or capsule. The influence of naphthol is to change the odor and color of the stools.

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ANIMAL PARASITES AND THEIR EFFECTS.

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PHILADELPHIA.

TAPE-WORMS—CESTODA.

DR. FRITZ ZSCHOKKE, of Geneva,[1] in consequence of some difference of opinion between Küchenmeister and Braun, as to whether certain species of fishes are the source of the *Bothriocephalus latus*, was led to make experiments on the subject, as he says, in the locality long known as the chief seat of infection. With this object in view a number of zealous students of the university of that place submitted themselves to the trial. The supposed *Bothriocephalus* larvæ were sought among the fishes commonly brought to the market,—the pike, *Esox lucius;* the *Lota vulgaris;* the *Salmo umbla;* the perch, *Perca fluviatilis* and the *Coregonus fera.* A single larva was found in a considerable number of the pike, and it was likewise rare in the *Coregonus*, which was contrary to expectation, as these fishes especially are commonly supposed to produce infection. The other fishes, particularly the *Lota*, contained an abundance of the larvæ. The parasites occupied the wall of the pharynx, stomach, and pyloric cæca, the kidneys, the milts and roes, the dorsal muscles, the body cavity, and especially the liver.

Seven students swallowed some of the larvæ, and after a few weeks on taking medicine four of the number discharged mature tape-worms, which were recognized as the *B. latus.* Of these three complete worms ranged from 130 to 196 centimetres in length, with from 750 to 900 segments or proglottides. Of the infected students, two swallowed larvæ from the *Lota*, the others from the *Salmo.* Of those not infected one partook of the larvæ both from the *Lota* and *Salmo*, and the others the larvæ from the perch. The author considers the *Lota* to be the common source of infection in Geneva, where it is brought to the market in large quantities, is cheap and much liked. The liver, roes, and milts of the fish which are especially infected, when slightly cooked are regarded as a great dainty. He also incidentally remarks that cases of *B. latus* have considerably diminished in Geneva during the last thirty years, and in the last decennium have decreased to such an extent that a case is now rare. On the other hand, the *Tænia saginata* has proportionately increased and is now the dominant tape-worm of the place.

In regard to the pike, which Küchenmeister declares never to be the intermediate host of the *B. latus*, Prof. Leuckart refers to the experiments and statements of Prof. Braun, who states that in the locality of Dorpat, long since known as a productive source of the *Bothriocephalus*, the pike is infected, both in flesh and viscera, with larvæ, which when swallowed by man, and also by the dog and cat, develop before a month elapses into mature tape-worms with all the characteristics of *B. latus*. Dr. Ferrara[3] further reports that having assured himself that he was not infected with tape-worms, on the 5th of July swallowed three reputed *Bothriocephalus* larvæ from pikes of Sicily. On the 20th of August he found eggs in the fæces, and on the 30th, after taking the proper remedy, he discharged three worms respectively 333, 340, and 480 centimetres long, which on reference to Prof. Leuckart, were pronounced to be the *B. latus*.

Dr. E. Parona, of Lombardy,[4] found the larvæ of *Bothriocephalus* in the flesh and viscera of the pike, *Esox lucius*, and of the perch, *Perca fluviatilis*. The larvæ were fed to four dogs and were also swallowed by a man, and in all cases they gave rise to the development of complete tape-worms, regarded as the *B. latus*. The author regards the experiments as the more conclusive from having been made in a locality where the latter parasite rarely occurs in man.

E. van Beneden[5] relates the case of a girl in Belgium affected with *B. latus*, which is considered remarkable in view of its rarity there, while it is so frequent in the neighboring country of Holland. The author, however, thinks it is more common in Belgium than is generally supposed, but is not distinguished from other tape-worms.

M. Braun, of Rostock,[6] referring to differences between the larva of *Bothriocephalus* of the pike in the locality of Dorpat, Russia, and that in a variety of fishes of Switzerland, especially the *Lota*, quotes Dr. Zschokke, who from comparisons, notwithstanding the variations, regards them as identical in species.

Dr. Roux,[7] of the Cantonal Hospital at Lausanne, reports the case of a young woman who, after the use of two six-gramme doses of extract of male fern, discharged ninety individuals of *B. latus* ranging from sixty to two hundred and thirty centimetres, exclusive of numerous headless specimens. The patient was healthy and, except occasional headaches and vivid dreams, presented no morbid symptoms. The infection is attributed to the use of fishes from Lake Leman.

Dr. Foot[8] says the *Tænia saginata* is entitled to be called the common tape-worm. The mature segments of this species are disposed to wander on the outside of their host, which is rarely observed in those of *Tænia solium.* Acetic acid, by rendering the segments translucent, brings into view the uterus, which in *T. saginata* has many branches, but comparatively few in *T. solium.*

Prof. B. Grassi,[9] of Catania, Sicily, after observing tape-worm eggs in the fæces of a little girl of Milan, and later of two children of Catania, on the failure of kousso in the former prescribed for the latter six grains of the ether. ext. of male fern in half a glass of gum-water. This treatment was followed by the discharge from each patient of several thousand small tape-worms, varying from eight to fifteen millimetres long, and recognized as the *Tænia nana*, previously found but once, by Bilharz, in Egypt, in a child who died of supposed meningitis. The middle and posterior segments of the worms were yellowish, due to the contained eggs. The corona of hooks numbered about twenty-seven; and the genital organs resembled those of *Tænia flavopunctata* as figured by Leuckart. With the aid of M. Calandruccio, many experiments were made with the eggs, but only with negative results. They were fed to various animals, as a sucking kid, a pup, chickens, a guinea-pig, many different myriapods, many larvæ of various flies, roaches, fleas, bugs, and lice. A man, also, who swallowed a pill full of fresh eggs, produced no tape-worms. More satisfactory results followed the medical treatment of the cases, which were marked by severe nervous symptoms, epileptic attacks without loss of consciousness, dullness of intellect, melancholy and insatiable appetite, all of which symptoms almost completely disappeared after the expulsion of the parasites. Later the author refers to six additional cases, mostly with a small number of worms and slight nervous symptoms, but which in one case were very severe. Still later Prof. Grassi says the *T. nana* is much more common in Sicily than other species, and he and M. Calandruccio have seen seven additional cases, all accompanied with considerable intestinal disturbance and reflex symptoms.

The prevailing theory that tape-worms require to pass the several stages of their existence in different animals, from the later researches of Prof. Grassi, would appear not to be without exceptions. Numerous experiments were made by this author and M. Calandruccio, with the view of ascertaining the intermediate hosts of the dwarf tape-worm, *Tænia nana*, and the nearly-allied *T. murina* of the rat, *Mus decumanus*. As Küchenmeister and Leuckart had indicated the meal-worm, *Tenebrio molitor*, to be the intermediate host of the *Tænia murina*, numerous attempts were made to develop both this species and *T. nana* through the larvæ of that insect, but always with failure. Some thousands of the larvæ, from houses infested with rats having the tape-worm, and where persons affected with the dwarf tape-worm lived, were examined, and only in a single instance were two Cysticercus-like scolices found; and these swallowed by a man produced no result. Numerous observations and experiments were made by feeding the eggs of the two tape-worms to many arthropods, worms, and mollusks, but all with negative results. Finally, experiments proved that the *T. murina* requires no intermediate host, and there is reason to believe that this is also the case with the *T. nana*. Albino rats were chosen as the subjects of experiments frequently repeated. In all cases, when these were furnished with ripe proglottides with eggs, tape-worms were produced, often more than a hundred together; while others, kept under the same circumstances without the proglottides, remained unaffected. The author traced the development of the mature worms from the eggs given to the rats. It was found that the eggs require to pass through the stomach before they are developed into the mature worms in the intestine. Similar experiments with six persons did not succeed in the production of the *T. nana;* but a boy who was previously unaffected, and was employed from time to time to seek for the worm in the fæces of another who was infected, became himself infected, and after treatment discharged fifty worms. Experiments with the *Tænia elliptica* of the dog, which, according to Leuckart and Melinkow, has for its intermediate host the louse, *Trichodectes*, of the same animal, likewise failed in positive results. The latter accord with an observation of Dr. A. Lutz,[9] of Brazil, who had a dog greatly infected with *T. elliptica*, but on whom no lice could be detected. Dr. Grassi concludes that *Tænia nana*, *T. murina*, and *T. elliptica* may undergo direct and complete development in a single host.

The latest researches of Prof. Grassi,[9] in association with M. Calandruccio, render it probable that the *T. murina*, very common in the rat of Sicily, is a mere variety of the *T. nana*. If such is the case it becomes a question whether the rat may not be the ordinary source of infection of the latter in man. The author continues that it may be said the *T. nana* is more common in Sicily than the other species of human tape-worms. He refers to fourteen additional cases, mostly in children, to whom the investigations were chiefly confined. The parasite is easily diagnosed by microscopic examination of the fæces; but it should be remembered that it is possible that eggs may be absent even when there are numerous worms, so that it is important to make repeated examinations at intervals of several days. The number of worms in an individual varies greatly,—forty or fifty, even to four or five thousand. It is further to be noted that a case may be greatly infected and yet appear perfectly well; thus a girl of four years, who was strong and active and occasionally was troubled with belly-ache, was infected with thousands of the worm. Costiveness, alternating with diarrhœa and severe belly-ache, are the most frequent symptoms, not infrequently accompanied by the reflex symptoms of helminthiasis, as epileptic disturbance, etc. As concerns the treatment, the male fern is a safe remedy, but it may be necessary to repeat it after a couple of weeks.

Dr. E. Comnini, of Varese,[10] reports the case of a boy who suffered with severe epileptic fits and discharged large numbers of the *T. nana*. After treatment with the extract of male fern and the expulsion of the worms, the fits ceased, but returned after three months, and with them eggs of the *Tænia* in the fæces. Later he observed another case of a girl who suffered with gastro-enteric symptoms accompanied by asthma and dyspnœa. In similar nervous disturbances the author recommends a careful examination of the fæces.

Dr. E. Parona[11] reports the case of a little girl of Varese, Lombardy, who discharged four small tape-worms which he supposed to be the *T. flavopunctata*, previously only noticed in the United States; but Prof. Grassi has determined them to be the *T. leptocephala*, which ordinarily infects the domestic mouse.

Dr. Krüger, St. Petersburg, relates the case of a child troubled with gastro-intestinal disturbance and fever, who passed some proglottides, and after treatment with kamala, portions of a tape-worm together a metre long, which proved to be *T. elliptica*, or *cucumerina*, common in the dog.

Dr. H. Krabbe[12] gives an account of his observations of three hundred cases of tape-worms occurring in Denmark, more than half of which were in Copenhagen. Eighty-five were in males, the others in females. *Tœnia saginata* occurred in 190 cases, always solitary; *T. solium* in 77 cases, multiple in thirteen up to ten worms; *T. elliptica* or *cucumerina* in nine cases, double in three; and *Bothriocephalus latus* in 25 cases, multiple in five up to fourteen worms. In one case *T. solium* and *B. latus* occurred together. It is especially noteworthy that *T. elliptica*, a common tape-worm of the dog and cat, occurred only in infants under one year, and in which no others were found. As the larval condition of this species is reputed to live in the louse of the dog and cat, the infant may be infected like the latter by swallowing the louse, or the eggs of the worm in its food or otherwise. The remedy employed in most of the cases since 1880, and with good effect, was the ethereal extract of male fern followed by a laxative.

In answer to an afflicted correspondent,[13] five different physicians recommend for the treatment of *T. solium* the oil of male fern. Dr. F. A. A. Smith states that while in South Africa he treated many cases, invariably with success. The first day keeping the patient on low diet of thin soup, he prescribed a drachm of the compound powder of jalap. The second day, on an empty stomach, he gave two or three drachms of oil of turpentine and one drachm of oil of male fern in emulsion; and after half an hour, a dose of castor-oil. He expresses the belief that every native is infected with tape-worm; and this is confirmed by another physician, M. C. Hobson, long a resident of the country. Another physician, G. N. Harris, recommends an Indian remedy "bao-birang," the powdered seeds of *Embelia ribes*, which he adds is largely imported into Germany, where it forms the chief ingredient of several patent remedies for tape-worms.

Two physicians, Whiteford and Boetz,[14] give their experience in the treatment of tape-worm in two children, eighteen months and three years of age, in which they found the extract of male fern an effectual remedy.

M. Wannebroucq[15] had occasion more recently to treat ten cases of *Tænia saginata*, in all of which he succeeded in expelling the worm by means of the fresh ethereal extract of male fern, given in the dose of four or five grammes. It is important that the medicine should be fresh, in which state it proved effectual in several patients in whom other tænifuges and the old extract had failed.

In the treatment of tape-worm Dr. K. Bettleheim,[16] of Vienna, recommends the following pills coated with keratin: ℞. Ext. filicis maris æth., ext. punicæ granati, āā 10 grammes; pulv. jalapæ 3 grammes.—M. Make seventy pills with keratin coating. The keratin prevents solution of the pills in the stomach and consequent emetic effects. The dose is fifteen to twenty pills given on the day of fasting, which is preceded by a cathartic. The remaining pills are given on the following day within two or three hours; and when necessary followed by a purgative.

Dr. Buckingham reported in the Boston Society for Medical Improvement that after failing with a number of remedies in the treatment of that most obstinate of the tape-worms, the *T. saginata*, he had succeeded in expelling it by means of pelletierine. He gave three decigrammes of the sulphate with the addition of tannic acid, recommended as preventing the speedy absorption of the former. A cathartic directed to be taken half an hour afterwards, in consequence of much nausea was postponed for an hour and a quarter. Referring to the usual manner of prescribing pomegranate, he points out that observations indicate that it should be given in one or two doses, and not in small ones repeated over a considerable time.

Dr. I. Harris,[18] of Tripoli, Syria, informs us that one-fifth of the inhabitants are affected with tape-worm, due to the consecutive habit of eating raw meat and defecating in the open fields. He treated fifty-five cases successfully with the ethereal extract of male fern; mostly two drachms of the remedy.

Dr. Numa Campi,[19] an Italian physician, gives his testimony to the use of thymol for tape-worms, and says it is both a tænicide and a tænifuge. Its action is speedy, and with the exception of a depressing effect easily counteracted by ordinary stimulants, it produces no disturbance of the bowels. From its efficiency in expelling the beef tape-worm, which is the most resistant to the action of anthelmintics, it is inferred that it will expel all other kinds. Dr. Campi prescribes half an ounce of castor-oil in the evening, with abstinence from food; the next morning two drachms of thymol are divided into twelve doses, of which one is taken every quarter of an hour; and half an hour after the last dose of the former, another half ounce of castor-oil is given. This is usually followed by the expulsion of the dead worm.

Dr. Koriander,[20] a Russian physician, recommends naphthalin as a tænicide. For children from one to three years, two to thirty grains; for adults, twenty to ninety grains daily.

Chloroform is also stated to be an effective remedy for tape-worm. Mr. Thompson[21] successfully employed one drachm by weight, with one ounce of simple syrup, given in three doses, at intervals of two hours, taken fasting, and with castor-oil to follow.

Dr. H. A. Veazie[22] relates a case of tape-worm in a boy of three years cured by feeding him on pumpkin pie and dried pumpkin-seeds.

Dr. N. Vakulovsky,[23] of Cronstadt, treated a woman with *T. saginata*, which was expelled by a strong decoction of the dried flowers of mignonette. He does not state whether it was the *Reseda odorata* or the *R. luteola*, of which the latter is an old popular remedy in Russia for tape-worm.

PERNICIOUS ANÆMIA ASSOCIATED WITH BOTHRIOCEPHALUS LATUS.

Dr. Runeberg,[24] in charge of the clinic at Helsingfors, reports nine deaths from progressive pernicious anæmia from 1878 to 1883; but in nineteen cases from that time to September, 1886, only one death, which occurred on the day of presentation at the clinic. The others were all cured after the diagnosis of *Bothriocephalus latus* and its expulsion by appropriate remedies. The author had a similar experience in Finnland, and likewise Dr. von Reyer in the Baltic provinces; so that it was inferred that the Bothriocephalus, and probably other intestinal parasites, are to be viewed as the cause of the anæmia. This view is questioned by

Drs. Biemer, Quincke, Immermann, and Heller, who consider that these cases have no relation to the morbid condition described by Biemer as progressive pernicious anæmia. Heller added that he had frequently seen numerous *Bothriocephali*, up to seventy-eight in a single individual, without any detriment to the general health.

Dr. G. Reyher[25] reports his experience in thirteen cases of progressive pernicious anæmia, which malady he had previously regarded as necessarily fatal. All the symptoms of the affection were present and the usual treatment proved of no service, until after segments of *Bothriocephalus* were passed, when the expulsion of the parasite by the extract of male fern was followed by recovery of the patients. The author refers to the similar condition of miner's anæmia, caused by the *Anchylostomum duodenale*, and intimates that other cases may be due to parasites. With these facts in view it becomes important in all similar cases that the patient's discharges should be carefully examined for the ova of worms, when if found the treatment is obvious.

Dr. N. Schapiro,[26] of St. Petersburg, after referring to the observations of Runeberg, Reyher, and others on pernicious anæmia with *Bothriocephalus*, relates the case of a boy who suffered with all the symptoms of the disease. He was prescribed a good diet with tonics, but under this treatment showed no improvement, but rather grew worse. His blood exhibited a great diminution in corpuscles; and an examination of his discharges showed numerous eggs of *Bothriocephalus*. The extract of male fern caused the expulsion of large masses of worms, but the condition of the patient seemed to be aggravated. The temperature arose to a high febrile degree, and delirium with a typhus condition set in. After several days a crisis occurred, the fever subsided, the other symptoms declined, and the patient rapidly recovered. Prof. Eichwald remarked in a clinic on the case that he had repeatedly observed a febrile state occurring after the use of an anthelmintic previous to the discharge of the dead worms. He has also observed the same condition after the spontaneous death of tape-worms, which at the time produced obstruction of the bowel.

CYSTICERCUS.

M. Karenski[27] states that from November, 1884, to February, 1887, in 8500 patients he had observed *Cysticerci* nine times; six in females and three in males; twice in the mucous membranes, thrice in the skin, and four times in the muscles. They were always single, and in the latter cases in abscesses. One of the patients also had a *Tænia;* and in another, the mother of a nursling with the *Cysticercus*, had the *Tænia.* These two cases would appear to favor the idea that the presence of the latter led to the infection of the former. M. Virchow says that statistics do not resolve the question of the origin of infection. There is no proof that the eggs or proglottides of *Tænia* in the intestine will give origin to *Cysticerci* in the same individual unless they pass the pylorus, which can only happen through violent efforts of vomiting, of which he had nevertheless seen an example.

Prof. L. Hirt[28] gives a detailed and interesting account of a case of *Cysticercus* in the spinal cord,—probably the first of the kind noticed. The patient, a man of 66 years, of healthy family, entered the poor-house in July, 1885. In December he could stand or walk with difficulty, and preferably remained in bed. After some weeks he was seized with vomiting in the morning, and subsequently he had ptosis and paralysis of the left abducent nerve. The left pupil was decidedly larger than the right. Later came on complete incontinence of urine, failure of nutrition, and finally death in June, 1886. The spinal marrow appeared normal, but in a number of places beneath the pia mater there were clear vesicular bodies, on the average from 0.75 to 0.5 centimetre long, which proved to be *Cysticerci;* altogether in the dural sheath from fifteen to twenty.

Dr. McReddie,[29] of Glasgow, referring to the statement of Prof. W. von Zehender that it is noteworthy that hookless *Cysticerci* (*T. saginata*), such as infect cattle, have never been found in the human body, says that in India on two occasions he found hookless *Cysticerci* in the intestines of natives of low caste, who use the flesh of cattle when they can afford it.

Dr. Gavoy,[31] in the hospital of Limoges, examined a man aged 42 years, a pork-butcher, who died after two years' illness. In the brain he found *Cysticerci*, at the bottom of the fissure of Rolando, the border of the Sylvian fissure, in the gray substance of the ascending frontal and parietal convolutions, and on the inner surface of the corpus striatum. These, by comparison, proved to be identical in character with the scolex or head of two individuals of *T. solium* discharged by young men at the military hospital of Versailles. Compared with some hundreds of *Cysticercus* from pork-measles, they proved to be different in the latter having no trace of pigment, and in having the hooks longer and less curved. He concludes, contrary to the general opinion, that there is no identity between the *Cysticercus* of measly pork and the head of *T. solium.* This view appears to be confirmed by Dr. Adolphe Hannover,[32] who, in the International Medical Congress of Copenhagen, expressed doubt as to the identity of the *Cysticercus* of the human brain with that of the *C. cellulosæ* of the hog. He says they show anatomical differences which alone are not important, but taken together have considerable weight. In the human *Cysticercus* the rostellum is more conical, in that of the hog more vaulted; and in this the larger hooks are longer and the smaller ones more curved.

Dr. Segay,[33] in referring to the facts as stated by Dr. Gavoy, remarked that they were not in accordance with the view generally admitted, and which had been so well exemplified by observations. While the presence or absence of hooks would be regarded as a good distinctive character of a species, it is not so with a difference in the length and curve of the hooks, and in the presence or absence of pigment. Of the Algerine tape-worm, reported by M. Redon as affecting the soldiers and Arabs in South Algiers, where there are no hogs, R. Blanchard says it refers to the unarmed tape-worm, probably a variety of the *Tænia saginata.*

Dr. Carles[34] informs us that in the Pyrenees hogs are mostly killed in the cold, dry weather of January and February, and the pork is preserved simply by drying. From the consumption of this, unsalted and uncooked, the people are largely infected with tape-worms; and it is asserted that in some cantons more than a third of the inhabitants harbor these parasites.

Dr. Armaignac[35] remarked that he now only met with the unarmed tape-worm in Bordeaux, where the armed one had become rare, though many butchers themselves eat measly pork. Dr. Mauriac observed that he also for a long time had only met with the unarmed tape-worm; but further said that if persons used measly pork with impunity it was only after it had been well cooked.

ECHINOCOCCUS TUMORS.

Dr. E. Frick[36] records two successful cases of the opening of *Echinococcus* cysts, with the removal of the parasites. In one case, that of a child, the tumor involved the liver and right lung; in the other it occupied the liver.

Dr. Leopold Landau,[37] of the University of Berlin, read an interesting paper describing four cases of *Echinococcus* which were removed with the cure of the patients. Three of the cases were females of 30 years of age or upwards, and the tumor occupied the liver, and in one of them a tumor was also in the left thigh. In the fourth case, a man of 34 years of age, who suffered for ten years, there were three distinct groups of tumors, which did not communicate. The largest cyst, occupying the liver, when opened, discharged six litres of *Echinococci*, and the remaining cavity was larger than a man's head. Another cyst under the ribs occupied the omentum, and the third was situated in the parietal peritoneum of the ileum, or in the mesocolon of the sigmoid flexure.

Dr. Arnaldo Viti,[38] of Sienna, relates the case of a man, aged 49, who died of a cerebral affection. He had an intrapelvic tumor since childhood, which had never troubled him, and of which the character had not been diagnosed. The autopsy revealed an enormous hydatid cyst, which occupied the recto-vesical space, mounting towards the umbilicus.

Dr. Tillaux[39] says the hydatic tremor, considered a reliable diagnostic sign, is due to the impulse of the daughter sacs upon one another in the absence of liquid in the parent sac. When the sac contains liquid the daughter sacs swim, and do not yield the tremor.

Dr. H. Vierordt,[40] in a treatise on multilocular *Echinococcus*, gives a synopsis of 75 observed cases, and, in addition, 4 new ones, in the clinic of Tübingen. Of these, 29 occurred in Bavaria, 21 in Switzerland, 18 in Wirtemberg, 7 in Austria, 4 in Russia, 2 in Prussia, 1 in Baden, and 1 in the United States. The greater number were in females, and generally in the liver, especially the right lobe. The diagnosis is difficult and the prognosis very grave.

FLUKE-WORMS—TREMATODA.

Bilharzia hœmatobia.—Mr. W. K. Hatch,[41] surgeon at Bombay, after having observed closely a dozen cases of the fluke-worm, *Bilharzia*, which appears here to be more common than formerly, especially among Mussulmen who have made the pilgrimage to Mecca, gives the following points of diagnosis, which he says may be of use to army surgeons, now that invalids are constantly returning from Egypt. Pain during micturition, with stoppages of short duration, and the passage of a few clots of blood. The urine is seldom discolored, but a few small clots and fragments of fibrin will be seen, especially on standing. The latter usually contain ova, but the former not so frequently. If there are no such fragments, a catheter may be passed, when shreds with ova will be usually brought away in the eye of the instrument. In hæmaturia due to *Filaria sanguinis* the urine is much discolored, and yellow, jelly-like clots are often present, while there is no pain and rarely stoppage during micturition. He adds that the patient with *Bilharzia* is often robust and not the least anæmic unless the affection has lasted for years. The time between infection and the passage of blood and other symptoms may be very short.

Mr. E. B. Hartley,[42] surgeon in King Williamstown, Cape Colony, referring to the above notice, remarks on the same affection in the latter place. For some years he met with occasional cases of hæmaturia among the soldiers, but recently he has treated numerous cases, in boys from ten to eighteen years, clearly due to *Bilharzia*. The affection appears to be common, in the town, in European lads, but no cases occurred in girls. He observes that the disease is not common in the higher altitudes of the colony, and is rare among adults. It lasts from three months to three or four years, but appears to produce no permanent damage. As a remedy, injections into the bladder he has found useless; and such is also the case with many remedies taken by the mouth. Preparations of iron he found most useful, especially "Bland's Pills" (ferri sulph., potas. carb., āā gr. ijss).

Surgeon C. H. Eyles,[43] Gold Coast Colony, referring to the occurrence of *Bilharzia* with hæmaturia, in North and South Africa, also reports four cases which came under his observation on the Gold Coast: three in natives, and one in a European who became infected in Egypt.

Dr. A. Napier,[44] of Crosshill, relates the case of a man, suffering from hæmaturia due to *Bilharzia*, who had resided eight months in Egypt. The patient stated that he believed that one-half of the inhabitants living along the Sweet Water Canal suffer from the affection, due to drinking the foul water of the canal.

THREAD-WORMS—NEMATODA.

Prof. B. Grassi,[45] of Catania, informs us that one of his students, after repeated examination of his fæces during six months without finding eggs or worms, on the 27th of June swallowed a number of eggs with embryos of *Trichocephalus*. On the 24th of July he first found eggs of this parasite in the fæces, from which it is concluded the embryos previously swallowed had reached mature development. The same student swallowed numerous eggs with the embryos of the *Ascaris lumbricoides*, but without the slightest result; and this was likewise the case in a repetition of the experiment. It proved otherwise with a boy of seven years of age, who after due examination to be certain that he was not affected, at the end of September was given a pill containing upwards of 150 ripe embryos of *Ascarides*. For a long time no eggs appeared in the fæces; but on the last day of November they were numerous. January 8th the author reports that the boy for several days, without having previously exhibited a symptom of helminthiasis, discharged 143 worms from 180 to 230 millimetres long. In that locality it is rare to see so many from the same person. During the experiment all other means of infection were avoided, from which it is inferred that the boy was infected by the embryos swallowed in the pill. The experiment with the eggs of *Trichocephalus* with another young man succeeded with like results. From these experiments it is evident that both the *A. lumbricoides* and the *Trichocephalus dispar* are directly developed from the embryos of the eggs swallowed by the host.

Dr. Laboulbene[46] says the water we drink is the ordinary vehicle of the eggs of *Ascarides*, which according to his researches undergo direct development to maturity when swallowed. He adds that the increasing custom of filtering water explains the less frequent occurrence of the *A. lumbricoides*.

Dr. Adolph Lutz,[47] of Sao Paulo, Brazil, refers to the researches of Prof. Grassi, and gives a sketch of his observations in practice, which go to confirm the view that the infection of both *Ascarides* and *Trichocephali* occurs through swallowing the eggs with the food which has been in contact with dirt or earth infected by pigs or men, or by drinking unclean water infected in the same way. Children are much more liable to infection when creeping about on all-fours among the dirt. Prof. Leuckhart, in commenting on the observations of Dr. Lutz, as well as the experiments of Prof. Grassi, appears to be convinced of their conclusiveness as relates to these parasites.

Dr. De Grange[48] gives an account of the case of a colored child of five years of age, neither unhealthy nor thin in appearance, but with a ravenous appetite and itching of the nose, who after the use of santonine and calomel, followed by castor oil, discharged nearly a quart of worms, *A. lumbricoides*, of which there were 145 ranging from nine to fifteen inches.

Dr. Kartulis,[49] of Alexandria, Egypt, relates a remarkable case of a man aged 30 years, well built and muscular, admitted into the hospital on the 30th of July. He was weak and had little appetite, and complained of pain and pricking in the gastric and hepatic regions. The liver was very large and painful on pressure. On the 28th of July he passed several worms, *A. lumbricoides*, and, after taking santonine and calomel, he had several passages with worms. Profuse diarrhœa set in, and on August 4th he died. In post-mortem examination the small intestine was found filled with 120 worms; the large intestine contained about 20 and the same number occupied the stomach, and again the œsophagus and pharynx. Others had penetrated the gall-ducts and liver; thus 3 occupied the common bile-duct, 3 the hepatic duct, 5 the gall-bladder, and 60 the liver. This organ was greatly enlarged and filled with abscesses along the course of the worms. No eggs were found in the liver. The other organs appeared sound, but were deficient in blood.

Dr. S. Gulichnelli[50] reports the case of a child, with supposed meningitis due to exposure to the sun, who was seized with fits and fever, accompanied by bilious vomiting, nervous symptoms, forced extension of the head, rigidity of the muscles of the neck, strabismus, etc. Santonine was given for constipation, when a hundred *Ascarides* were discharged, and the child rapidly recovered.

Dr. G. Schlemmer[51] relates the case of a patient, threatened with abortion, who was entirely relieved of the symptoms after vomiting a worm (*A. lumbricoides*) about eight inches long.

A case is related in Calcutta[52] of a Hindu female who suffered from dysentery which yielded to none of the usual methods of treatment, but recovered after the discharge of eighteen round-worms (*A. lumbricoides*). The writer says the case taught him a lesson fully confirmed by a subsequent large experience in the Eden Hospital, Bengal. When in any disturbance of the digestive tract, or in reflex phenomena arising therefrom, and when the usual treatment for dysentery, diarrhœa, convulsions, etc., failed, santonine, he found, in a good proportion of cases, the proper remedy.

Dr. Beaven Rake,[53] of Trinidad, relates the case of a Hindu child aged five years, who died of asphyxia. A living round-worm (*A. lumbricoides*) three inches long was found lying above the the epiglottis. No worms were found elsewhere.

According to the observations and experiments of Dr. Coppola,[54] while santonine does not kill *Ascarides*, but at first quickens their movements, it finally renders them unable to resist the peristaltic current. He therefore recommends the use of a cathartic one or two days after the use of the former. As a substitute, equally effective and less disposed to produce unpleasant effects, he recommends santoninoxin, derived from santonine.

Dr. Sydney Martin[55] says the complete cure of thread-worms (*Oxyuris vermicularis*) is often very difficult. Where ordinary injections fail, he has found rhubarb in small doses an effectual remedy. The difficulty of curing these worms may be illustrated by the case of a patient in Birmingham,[56] aged 40 years, who appeals for relief, stating that he has suffered from them more or less since childhood, and in the last three or four years to an almost insupportable degree. Treatment under the direction of Dr. Cobbold and all others has failed to give relief.

Brigade Surgeon A. Porter,[59] Madras, referring to the statement of Dr. McConnell, of Calcutta, that the *Anchylostomum duodenale* is as common among the natives as the *Ascaris lumbricoides*, says that in nearly 1000 autopsies in Berar and Madras, while the latter was present in quite half, he had never seen the former but once. This case, a man aged 50, was admitted into the hospital on February 26th with diarrhœa and anæmia. On March 7th he died, and in the autopsy several hundred of the *Anchylostomum* were found adherent to the mucous membrane of the duodenum, which otherwise appeared healthy.

Calundruccio[58] notices four cases of anæmia in Sicily, due to the *Anchylostomum*. For this affection thymol, or the ethereal extract of male fern, is recommended.

From the observations and experiments of Dr. Meyers,[59] in South Formosa, it would appear that the *Filaria sanguinis* passes its intermediary stage in a particular species of mosquito, found in Amoy, but not in the former locality. He supposes that the remarkable periodicity exhibited by this parasite, remaining in the lymphatic system during the daytime and appearing in the blood at night, to be due to the greater proportion of oxygen at this time, which attracts it into the circulation. While he agrees with Dr. Manson in attributing elephantiasis to the *Filaria*, he considers that the disease is subsequently perpetuated, without its influence, as a condition of malnutrition.

Dr. S. Mackenzie,[60] of London, described the case of a gentleman from the West Indies, who had suffered from chyluria, off and on, since 1876, and which probably had originated much earlier. The urine passed was bright chocolate colored, but after standing, from the subsidence of the blood-corpuscles, it assumed a cream-white color. In the blood taken at night the *F. sanguinis* was always present, but was absent in that taken during the daytime. The author referred to the mosquito as the intermediary host of the parasite, and said it had been clearly demonstrated that it was a particular species which was alone capable of nurturing the parasite. He further described a mematode worm, which had been found in the urine, in two cases, and had been mistaken for the *Filaria sanguinis*, but belonged to a species of *Rhabditis*.

Dr. Bulgakoff, of Russia, relates the case of a lieutenant, affected in the right leg with the *Filaria medinensis*, supposed to have been contracted through drinking marsh water while traveling from Buchara to Merv.

Prof. B. Grassi,[64] of Catania, describes a supposed new species, with the name of *Filaria inermis*. One of nine and one-half centimetres was extracted from a tumor of the conjunctiva of a woman in Catania; another of eleven and one-half centimetres from the eye of a man in Milan; and a third of five and one-half centimetres from the eye of an ass in Pisa. The relation of this worm to the *Filaria loa* of western Africa is uncertain.

TREATMENT OF INTESTINAL WORMS.

Eillard[62] says that whenever a remedy for worms is given, a purgative should also be used, not only to expel the parasite which has been killed, but also to prevent its absorption. The oleaginous purgatives are recommended, as giving the best results as adjuvants to vermifuges. Several of the formulæ, among those recommended, are the following:—

For tape-worms:

℞ Pumpkin-seeds, cleaned and pounded,	25 to 45 grammes.
Honey,	20 "

M. et ft. electuarium.

Sig.—Dessert-spoonful every half hour.

℞ Ol. æther. filicis mas,	3 grammes.
Tinct. vanillæ,	3 "
Syr. terebinth.,	25 "
Mucil. acaciæ pulv.,	2 "
Aq. destil.,	25 "

To be taken in one dose, with an equal quantity of milk. A dose of castor-oil to be taken a few hours later.

For *Ascaris lumbricoides*:

℞ Santonini pulv.,	40 grammes.
Sacch. albi,	2000 "
Mucil. tragacanthi,	180 "

To be made into tablets of five decigrammes each; each with one centigramme of santonine. Five to thirty tablets for a dose.

For *Oxyuris vermicularis*:

℞ Sodii chlor.,	40 grammes.
Aquæ,	200 "

To be injected by the rectum.

℞ Ol. theobromi,	4 grammes.
Acidi tannici,	1 "

To be used in the form of a suppository.

Attention has been called to the pink-root, *Spigelia marilandica*,[63] as an anthelmintic without a superior, being especially effective in the *Ascaris lumbricoides*. The most effective preparation is the fluid extract, of which the dose for children is five to fifteen drops, for adults one-half to one and one-half drachms.

Dr. M. Martinet[64] recommends the leaves of *Sida floribunda*, a malvaceous plant from the vicinity of Lima, as a mechanical vermifuge, the effects being dependent on exceedingly minute spines.

Dr. C. B. Marklay,[65] Peoria, Ill., refers to the oil of *Chenopodium anthelminticum* as having no superior for the expulsion of the round-worm.

TRICHINA AND TRICHINOSIS.

Of the recent occurrence of these in this country we have had the following notices:—

Dr. A. C. Kinney,[66] of Astoria, Oregon, states that fifteen persons who partook of sandwiches of uncooked pork sausage, on the occasion of a New Year's reception, were all more or less affected with the symptoms of trichinosis. *Trichinæ* were detected in the sausage. The symptoms of the affection resembled those of typhoid fever, and swellings were a marked feature. Recovery was very slow. After fourteen months, none of the patients died, except one, who shortly after the attack went to California and died there in an unknown manner. The favorable outcome, though apparently in a measure due to the small quantity of *Trichinæ* ingested, was thought to be the result of mercurial treatment, which probably killed many of the parasites in the intestines.

A case of trichinosis is reported by Dr. T. Gaertner,[67] of Pittsburg. A laborer attacked with the symptoms died. He was in the habit of eating raw ham. A piece from which he had eaten was found to contain active *Trichinæ*.

Cases were noticed at Steven's Point, Wisconsin,[68] of a man and his wife who died from eating pork known to be diseased.

In an article[69] entitled "Rats as an Agent in the Introduction of *Trichinæ* into Swine," the author states that his house, in proximity to some grain-cribs, became infested with rats. One of these, killed and examined, was found to be full of *Trichinæ*. Seven rats killed and thrown into the pig-pen were eaten by a brood-sow, which, fat at the time, became exceedingly emaciated and in six weeks died. All parts of the body, especially the intestines, muscles, and lungs, were literally filled with *Trichinæ*. Another hog, killed by a neighbor because he could not fatten it, was likewise found to be infected. Three cats which caught and ate the rats died. The author considerately expresses the opinion that it is criminal to leave the dead bodies of rats and mice unburied or unburned.

Dr. G. W. Furey,[70] of Sunbury, gives his experience of the first cases of trichinosis observed in Central Pennsylvania, in 1880.

A family of five persons was more or less affected with the characteristic symptoms due to eating infected pork. In all there was early languor, œdema of the face and eyelids, congestion of the conjunctiva and lameness in the orbital muscles, hoarseness, irritable fauces, difficult deglutition, cephalalgia and muscular pains, with especial lameness in the trapezius. In two only was there swelling of the limbs; in one profuse perspiration, in another gastric disturbance; in one, for a few days, colic, neuralgia, and diarrhœa; and two had considerable bronchial trouble. Fever, present in all, varied in degree and duration. The most marked of the cases under treatment was regarded out of danger after forty-two days. The chief remedy employed, and regarded as effective, was sodium sulpho-carbonate. Dr. Furey further refers to half a dozen cases of the affection in a family in Bethlehem, in 1855, of which the mother and two daughters died, and the others under the same treatment recovered. Mr. Eugene A. Rau, of Bethlehem, is mentioned as having demonstrated these cases to be trichinosis, and of his having made the most satisfactory preparations and photographs of the infected flesh of the patients who died. (This is fully confirmed by the present writer.)

In a notice of the occurrence of trichinosis in Zéland, Holland, Dr. J. Moens[71] reports that at Goes, a place of about 7000 people, he had observed sixty cases, considered to be this affection, especially characterized by painful swellings of the muscles of the face and limbs. Specimens of salted pork used were examined and found to be full of *Trichinæ*, and rats fed on portions died. From a patient several *Trichinæ* were extracted by the harpoon. Though the cases were grave, up to the time of the report only one had died.

An epidemic of trichinosis is noticed in Hamburg; and 72 cases are reported as having occurred from the 21st of August to the 15th of September, of which 9 died. Of 47 cases scrupulously studied, 14 were very severely affected, 13 moderately, and 20 lightly. The most prominent symptoms were œdema and muscular pains, digestive trouble and febrile exacerbations, and difficulty in swallowing and breathing. Twenty-four cases were traced to the use of sausages, hams, and pork. Since the epidemic the Hamburg inspectors discovered twenty-two trichinous hogs, which came from Denmark.[72] In a notice of trichinosis in Carthagena, Spain, up to the time of the report, twenty-three persons have died.[73]

Prof. F. Legge,[74] of the University of Camerino, Italy, in an autopsy of a man, observed *Trichinæ* in the muscles throughout. The man had been a patient in the hospital of the place, and among other symptoms had obstinate diarrhœa and muscular pain. His death was attributed to inanition. It is stated that this is thought to be the first case of trichinosis observed in Italy.

A case of trichinosis occurred at the hospital at Molembeck,[75] near Brussels. The patient was a German who frequently ate American bacon. He was admitted into the hospital one day and died the next. His voice was feeble, the pulse thread-like, the skin cyanosed, and he had profuse diarrhœa from which he had suffered for five days. *Trichinæ* were found in all the muscles except the heart, and also in the intestines. This is stated to be the first case which has occurred in Belgium. Dr. Crocq, one of the physicians who had charge of the patient, expressed the opinion that the infection was due to American bacon eaten seven years (!) before the appearance of the acute symptoms.

Dr. J. A. Close[76] points out a new method of detecting *Trichinæ* in meat. This consists in submitting the suspected substance to a mixture of pepsine and hydrochloric acid, and placing the solution in a conical precipitating glass. If the parasites are present they may be readily detected, in the precipitate, by the microscope.

Dr. Grawitz,[77] an assistant of Professor Virchow, says that in one-third of the cases of so-called muscular rheumatism, examined after death, *Trichinæ* have been found. In many cases the parasite appears to have existed for years.

Dr. R. O. Beard[78] insists on the importance of rest in trichinosis. Excessive restlessness or indulgence in exercise he considers to be responsible for the disturbance and reëmigration of the parasites. Authorities agree that the safety of the patient is subserved by the speedy encystment of the *Trichinæ;* therefore anything which causes the parasite to change its position is prejudicial to recovery. This suggests the importance of absolute rest and the use of sedatives to control nervous excitement.

PSEUDO-TRICHINOSIS.

Dr. Paul Hopp,[79] under this name describes an affection of unknown cause closely resembling trichinosis in its symptoms and results, without the presence of the *Trichinæ.* It is exemplified in the case of a young woman, previously in good health, where symptoms and the post-mortem examination by Professor Recklinghausen, are given with the most careful detail. The affection was an acute parenchymatous inflammation of nearly the whole muscular system.

ANIMAL PARASITES IN GENERAL.

R. Blanchard,[80] of Paris, enumerates as the parasitic worms of man 7 species of Tænia, 4 of Bothriocephalus, 11 Trematoda, 22 Nematoda, and 1 Acanthocephalus. Of *Tænia nana* he relates a case in a girl in Belgrade who discharged 250 worms.

H. Eulenberg[81] reports the result of the obligatory examination of the hog and its preparations in Prussia for 1885. In 4,421,208 hogs examined there were 2387 infected with *Trichina,* and 13,653 were affected with measles or *Cysticercus.* Of American pork examined 101 were trichinous. The number of people affected with trichinosis were 79, besides a family in Berlin. The cases were mostly of a light character, and only one death occurred. The number of measly hogs in the district of Berlin was 1 in 124; Potsdam, 1 in 262; Frankfort, 1 in 204; Oppeln, 1 in 114; Breslau, 1 in 228; Liegnitz, 1 in 258; Posen, 1 in 218; and, most unfavorably, Schildberg, 1 in 74.

J. Esser and W. Schütz,[82] in the annual report of 1884–5, of the examination of hogs in Prussia, in 1,390,027 found 5447 measly or affected with *Cysticercus,* 698 with *Trichina,* and 117 with *Echinococcus.* In three districts, Bonn, Breslau and Coblentz, in 21,883 hogs there were none with *Trichina* and altogether 43 with *Cysticercus;* in Liegnitz, in 248,813 hogs, 164 had *Trichina* and 1171 *Cysticercus;* in Oppeln in 291,162 hogs, 40 had *Trichina* and 2277 *Cysticercus;* in Merseberg in 361,851 hogs, 67 had *Trichina* and 179 *Cysticercus.* In Aurich in 13,392 hogs there were only 4 with *Cysticercus* and none with *Trichina.*

The excellent work of Prof. R. Leuckart on "The Parasites of Man and the Diseases which proceed from them," has been translated from the German into English, by Wm. E. Hyle, of Edinburgh.[83] Leuckart disproves the older notion that parasites only attack unhealthy animals and tissues, and shows that when any real connection between the unhealthiness of the host and the presence of parasites exists, the former is the effect and not the cause of the latter. The list of affections produced by parasites is long and varied, but mostly helminthic affections are not sufficiently characteristic to lead to certain diagnosis, unless an objective proof of the existence of parasites is obtained. In this view the importance of examining the excreta with the microscope, to detect the presence of eggs, is insisted upon.

LICE.

Dr. Vastanian,[84] of Constantinople, recommends for the lice *Pediculus capitis* and *Phthirius pubis* frictions with flannel dipped in a solution of two or three parts of salicylic acid in twenty-five parts of vinegar and seventy-five parts of alcohol. Dr. A. V. Meigs,[85] of Philadelphia, recommends for the same lice a solution of one grain of corrosive sublimate to one ounce of alcohol; a single or possibly two applications are sufficient for the former and two or three for the latter.

Dr. B. F. Greenough,[86] of Boston, recommends crude petroleum for the *Phthirius pubis;* and Dr. G. P. Thomas, of Alameda,[87] Cal., has found a single application of ether effectual for the same louse. (We may here say that if it is once understood that all insects, including lice, are quickly destroyed by the application of any fixed or volatile oil, physicians will see there is no necessity of employing remedies of a noxious character to the patient. The fat of mercurial ointment is probably more quickly active than the mercurial oxide.)

FLY PARASITES.

Dr. Ralph Matas,[88] of New Orleans, after relating the case of a man from Honduras, from whom he extracted in the gluteal region three Œstrus larvæ, in view of our prospect of increased relations with that country gives a general history of the Œstrus parasites which infest man. Among them are the bot-fly of the ox, *Hypoderma bovis*, and that of the sheep, *Œstrus ovis*, which exceptionally attack man. Several species of bot-flies of another genus, *Cuterebra*, or *Dermatobia*, while not peculiar to man, more frequently attack him in tropical America. Dr. Matas refers to Dr. LeConte's experience with the insect in Honduras. Dr. LeConte relates that while traveling in that country, several of his companions were much afflicted with the larvæ. They seemed to infest parts of the body not usually exposed; and it is supposed their eggs were deposited while the patients were bathing. They produce a swelling like an ordinary boil, in which at times, for a few seconds, an acute pain is felt. By applying heat, as of a lighted cigar, as near as the patient can bear, the worm becomes restless and the point of its body appears at a minute opening of the skin, not before obvious, when by violent pressure of the tumor the larva is forced out. Sometimes it is so small as to resist this mode of extraction; when, if tobacco be applied for a few hours, the worm dies, and may then readily be squeezed out. No inflammation or discharge follows, but the sac readily heals. (The present writer has in his possession nine specimens of the larvæ which were obtained by Dr. LeConte.)

Dr. Gustav Joseph,[89] of Breslau, gives a succinct history with his experience of "flies as pernicious animals and parasites of man." To the pathological condition of the stomach and intestines induced by the presence of the larvæ of flies, he gives the name of *Myiasis interna;* and to that caused locally, and thence sometimes internally, through the bites or contact of mature flies, *M. septica.* Though the eggs of flies deposited in cold food of various kinds are frequently swallowed, it is only in a few species that they are hatched, and the larvæ occasionally live for some time in the stomach and intestines. The larvæ are facultative parasites, or such as ordinarily live outside in stercoraceous, decaying, and fermenting or other matters, but which are also able for a time to live within living animals. These belong to the family of Muscidæ and never to the Œstridæ. They occasion more or less gastric and internal disturbance, with pain, vomiting, diarrhœa, and dysentery. The author gives an account of many cases observed by himself and others, of fourteen different species of larvæ, which were discharged living in the fæces, or by vomiting. Flies which do not bite, like the common house-fly, may convey infection, by sucking or even touching infected matter and transferring it to wounded surfaces. Biting flies may likewise convey infection in this manner, but also may induce more or less serious effects by means of their own poison. Of the biting flies of Europe, the author mentions the *Stomoxys calcitrans*, also common with us, the *Hœmatopota pluvialis*, *Tabanus bovis* and *T. rusticus*, *Chrysops* *cæcutiens* and *C. relictus*, the *Simulium columbaschense*, and many gnats or species of *Culex*.

In another article Dr. Joseph[90] gives an account of the occurrence and development of bot-fly larvæ in the subcutaneous tissue of man. The larva of the bot-fly of the ox, *H. bovis*, not unfrequently occurs as a parasite of man.

S. Calundruccio, of Catania, notices the case of a boy with a sore on his neck from which a larva was removed and proved to be that of the *H. bovis*, or bot-fly of the ox.

Dr. Voltolini[91] tells of two cases of boys who slept in a stable and were affected in the ear by the larvæ of a fly, *Sarcophila wohlfahrti*. One died from osteo-phlebitis extending into the skull; the other recovered. They were treated with injections of oil of turpentine, followed by the application of ice and acetate of lead. (The writer would suggest the use of any mild oil, as lard or olive-oil, instead of turpentine, as being equally destructive to the larvæ of all flies, without having the irritating effect of the latter.) Mr. Joseph Portchinski, of St. Petersburg, states that the majority of cases of "malum verminosum" are produced by the larvæ of the above fly, first indicated in 1768 by Dr. J. A. Wohlfahrt in "Observatio de Vermibus per Naris excretis."

Dr. Wacker,[92] of Landsberg, Bavaria, reports the case of a boy who, after attacks of nausea, passed two litres of the larvæ of a flower-fly, *Anthomyia cunicularis*. (The present writer once met with the case of a physician who, after symptoms of cholera morbus, vomited a number of larvæ of a flower-fly. He supposed he had swallowed the eggs on some cold boiled cabbage.)

PARASITIC AFFECTIONS OF THE EYE.

Professor Zehender,[93] of Rostock, before the Society of Ophthalmology, discoursed on the subject of parasitic affections of the eye. Von Nordmann appears to have been the first to observe entozoa in the eye. He discovered a thread-worm in the crystalline lens of the eye of an old man; and he further reported the case of a woman, with cataract, in whom the crystalline lens contained a worm, to which he gave the name of the *Filaria oculi humani*. No case of the kind is found in recent records. A *Filaria* is sometimes found in the conjunctiva in the negroes of western Africa. The *Echinococcus* is rare, and occurs in the orbit, but not in the eyeball. It produces an exophthalmia more or less severe. Intraocular *Cysticercus* is always that of the *Tœnia solium*, and seems more frequent in northern Germany than elsewhere: this is attributed to the more common use of pork. The *Cysticercus* may develop in the orbit, under the conjunctiva, in the anterior chamber, in the crystalline lens, but is frequently found in the vitreous humor or under the retina. In the last case it produces a detachment of the membrane and sometimes a perforation. Its movements often occasion violent pain and inflammation, with opacity of the vitreous humor. If opacity is not too great it may be distinguished by the ophthalmoscope. The treatment is difficult. If the parasite causes great pain and inflammation the eye should be at once enucleated; otherwise we may wait some time. Inflammation usually follows soon, necessitating operation. In several cases the *Cysticercus* has been successfully extirpated. De Graefe, of Halle, recently published statistics of forty-five cases: in thirty the immediate results were good; in the others the opacity of the vitreous humor or the mobility of the *Cysticercus* baffled the operation, and enucleation was the resource.

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DISEASES OF THE BLOOD AND SPLEEN · TUBERCULOSIS AND SCROFULA.

By JOHN GUITÉRAS, M.D., Ph.D.

CHARLESTON.

The contributions to this department of medicine during the past year have been very numerous and important. The fundamental laws that govern the pathology of the blood are, however, far from being established. The reason of this is that nothing definite is known of the life history of the blood cells. The attempts made to throw light on this obscure subject will be found in the following contributions to "The General Anatomy and Pathology, and the Chemistry of the Blood:"—

A. Mosso,[1] of Turin, has published elaborate researches into the necrobiotic changes of the corpuscular elements of the blood. He finds that none of the preserving media prevent the changes of the cells. The first evidences of necrobiotic changes are in his opinion the biconcave shape of the mammalian red corpuscles, and the distinctness of the nucleus in other vertebrates. Space does not allow us to follow all the morphological and chemical changes described by the author; but we must note as the most important, the formation of the blood-plates of Bizzozero out of the disintegrating red blood cells; the conversion of red blood cells into leucocytes and pus corpuscles, and finally the formation of a hyaline, gelatine-like substance which is the basis of the coagulation of the blood. The author calls attention to the facilty with which the alterations may be precipitated by rough handling and other means. He insists upon the remarkable difference of resisting power shown by corpuscles obtained at the same time from the same individual. Of clinical significance are his observations as to the general resisting power of blood obtained in different conditions of health, of disease, or experimentally induced pathological conditions. These different degrees of resisting power can be gauged by admixtures with salt solutions of varying strength from 0.75 to 0.40 per cent. The method consists in finding that degree of concentration which will produce a transparent mixture with the blood; a process which, if our author be correct, promises to rival in importance with hæmocytometry, and hæmoglobinometry. The spectroscopic reactions observed during the changes of the blood lose the characteristics of hæmoglobin and resemble those of the biliary coloring matters.

Many of the morphological changes of the red corpuscles correspond very closely, if they are not identical, with the appearances that have been supposed to indicate the presence of a peculiar micro-organism—the plasmodium malariæ—in the blood. Mosso concludes that they are the same. In a long series of observations on malarial blood made in the summer of 1886, the editor came to the same conclusion. The effect of oblique illumination especially led him to believe that the appearances in question were due to alterations in the cortical substance of the corpuscles. He was much impressed by the fact that a specimen of blood obtained immediately before death from pernicious malarial fever, showed very little deviation from the normal, and yet a histological examination of the cortical substance of the brain revealed the fact that the capillaries were engorged with pigment granules to an extraordinary degree. If these are contained in the micro-organisms, how can we explain the fact that all the plasmodia-bearing corpuscles should accumulate in these capillaries? From the point of view of a disintegration of blood corpuscles, on the other hand, the phenomenon becomes explainable. Mosso finds that a process of selection may take place in the capillaries, and that the least resisting corpuscles are detained, probably by their tendency to produce coagulation. It must be stated that the best authorities in this country, Councilman and Osler, favor the parasitic view of these phenomena.

The changes of the red corpuscles after withdrawal from the circulation have been also carefully studied by E. Marigliano.[2] He finds that syphilis, tuberculosis and chronic hepatic diseases, especially interstitial hepatitis, impair the resisting power of the red corpuscles; whilst fever exerts but little influence. The resisting capacity is not always in proportion to the oligæmia.

A. P. Fokker[3] expresses the opinion that the red corpuscles may continue to live indefinitely if removed with antiseptic precautions, and if placed in favorable nutritive media. The evidences of vitality are, the evolution of acid products, and the formation of bodies which the author calls hæmatocytes. These hæmatocytes are supposed to be living bodies, and our author believes that they may be transformed into bacteria.

Silbermann[4] finds in the blood of the newborn a large number of pale corpuscles (schatten) consisting of the stroma of the erythrocytes from which the coloring matter has been removed. He concludes that there is a condition of hæmoglobinæmia, which may give rise to pathological manifestations. He finds poikilocytes, increase of leucocytes, hyperæmia of the abdominal organs, a dark brown coloration of the liver, albumin and biliary coloring matter in the urine. With these evidences of destruction of red blood cells, there is an increase of fibrin ferment in the blood which may account for febrile disturbances.

The crystallization of the hæmoglobin within the red corpuscles is noted by Mosso[1] in slowly coagulating blood. This subject has been investigated by C. J. Bond[5] who found it to occur in the following diseases: in sapræmia more than in septicæmia or pyæmia. In fact the crystallization appears to depend more on conditions of toxæmia than infection. For this reason, in erysipelas the change will be noticed in blood drawn from the affected part, and may not occur in blood drawn from other regions. It is observed in cancrum oris and before the fatal termination in phthisis and in pneumonia, but not in anthrax, malaria, measles, smallpox, typhoid and scarlet fever. It is present in some cases of pernicious anæmia, and is very constant in leucocythæmia. Whenever the blood shows a tendency to crystallize it presents the spectrum of reduced hæmoglobin. Prof. Neucki,[6] of Berne, has succeeded in crystallizing reduced hæmoglobin. He does not believe that all animal pigments are derived from blood coloring matter, and he observes in this connection that the coloring matters of the hair and the choroid do not contain iron. Herman Lenharz[7] has found by spectroscopic analysis that the cause of death in poisoning by chlorate of potassium is the formation of methæmoglobin. The alkalinity of the blood has been observed by von Jaksch,[8] of Gratz, to be lessened in fevers, in uræmia, in destruction of the hepatic parenchyma, in leukæmia, pernicious anæmia, chlorosis, and poisoning by carbon dioxide.

The Migration of White Blood Cells.—T. Wharton Jones[9] insists upon the passive character of this phenomenon. Mosso[1] looks upon it as a diapedesis of red corpuscles more or less altered during and after the emigration. Rudolph Disselhorst,[10] in studying the influence of quinine, mercuric chloride, eucalyptol, etc., upon the migration of leucocytes, concludes that this phenomenon is influenced more by changes in the walls of the blood-vessels than by the activity of the leucocytes.

Hæmoglobinometres.—A. Sioling,[11] of Upsala, has made careful comparative estimates of the results obtained by the instruments of Gowers, Hayem, Malassez, Bizzozero, and Fleischl. He finds in the latter's hæmometre the greatest accuracy with simplicity of method. These conclusions are confirmed by Prof. Henschen.

The Arterial Pressure.—S. v. Basch[12] furnishes some interesting results obtained with the sphygmomanometre. This instrument, consisting of a water chamber in communication with a manometre, records the amount of pressure necessary to abolish the pulse in a given artery. The temporal is generally selected.

ANÆMIA AND CHLOROSIS.

Pathogenesis.—It is generally believed that the simple forms of anæmia are the result of diminished production of red blood corpuscles, whilst in pernicious anæmia we have increased destruction. This view is maintained by a recent investigator, Dr. Hunter.[30] Dr. Hénocque[31] has investigated the modifications of the activity of the reduction of hæmoglobin in the tissues in chlorosis. The energy of the oxygen consumption he finds much diminished in this disease. The anæmia, in fact, may disappear in cases of chlorosis, and still the impairment of the reduction of hæmoglobin may persist. In spinal irritation the reduction activity is increased.

J. Inglis Parsons[32] adheres to See's theory of chlorosis, that the cause of the disease is to be found in the rapid increase of all the tissues that takes place at the onset of puberty. He calls attention to the magnitude of this growth in the female as compared with the male. It has also been suggested[33] that chlorosis occurs in girls who menstruate prematurely, before the development of the breasts. Gräber,[34] of Munich, believes that chlorosis originates in an excessive alkalinity of the blood. An attempt has been made by Sir Andrew Clark[35] and Duclos[36] to connect chlorosis with an auto-infection or toxæmia resulting from the retention and decomposition of fæcal matters. Dr. Clark's paper contains an excellent description of a certain class of cases of anæmia, but is far from convincing when he attempts to establish fæcal toxæmia as the dominant morbid process in chlorosis. Certainly much importance must be attached to the violations of physiological laws practiced by women about the age of puberty. They, says Dr. Clark, tighten their waists, stint themselves in food, and often omit the daily solicitations of the bowels. Fæces accumulate. Ptomaines and leucomaines are produced. James Oliver[37] suggests that many cases of chlorosis may be due to alterations of the intestinal mucous membrane produced by the use of indigestible articles of diet, such as bird-seeds and even nut-shells.

Symptoms.—The estimation of the number, and the richness in hæmoglobin of the red corpuscles constitutes now an important element of diagnosis in anæmia, M. Mayet[38] proposes a new form of serum for the dilution of the blood in the operation of counting the corpuscles. It consists of a 2 per cent. salt solution to which enough sugar is added to raise the specific gravity to 1085. The difference in specific gravity places the white and the red corpuscles in a different focus in this solution. Hayem[39] has shown that the number of white blood corpuscles is increased in cases of neoplasms. Earnst Gräber [40] found in twenty-eight cases of uncomplicated chlorosis, that the number of red corpuscles was not diminished, the average being 4.482000. The average diameter he found slightly below the normal, and the average richness in hæmoglobin was reduced to 5.2 per cent.; the normal proportion, according to Hénocque,[41] being, in man from 13 to 14 per cent. Dr. Herman Sahli[42] calls attention to the modifications of the standard color solutions that are needed when the hæmoglobinometre is used with artificial light.

Ritter and Hirsch[43] conclude that a hyperacidity of the gastric secretion is not constant in anæmia and chlorosis, and that the gastric ulcers that occur in these diseases are not caused by the excess of acid in the stomach. Sir Andrew Clark[35] describes as follows the digestive symptoms of chlorosis: "The tongue is usually coated, indented and watery. The breath is tainted and occasionally fæcal The appetite is usually capricious. There is a sensation of sinking at the stomach between meals. The patients complain of pain at either flexure of the colon, generally the left, the pain extending through to the back. The bowels are either obstinately confined or inadequately relieved. The fæces, usually scanty, dark, hard and lumpy, occasionally consist of showers of scybalous masses imbedded in an offensive mucus swarming with bacteria." Dr. André,[44] of Toulouse, calls attention to a decrease in size of the liver which is present in chlorosis and absent in other forms of anæmia.

Duclos[45] and Durozier[46] insist upon the significance of cardiac and vascular murmurs in anæmia, whilst Apetz[47] gives statistical evidence to show that little importance can be attached to the venous hum in young persons, especially if it is heard only when the head is turned to the opposite side. 61 per cent. of non-anæmic individuals, between the ages of one and twenty years, presented a venous hum. These conclusions require further confirmation.

Barié,[48] following Peter, employs the surface thermometer to determine the existence of pulmonary tuberculosis in anæmia. The temperature of the second intercostal space near the sternum is usually 35.8° C., whilst in tuberculosis it rises to 36.3°, and even 38.8°. Again, in chlorosis the elimination of phosphates by the urine is diminished, and in disintegrating disease of the lung it is increased.

Treatment.—In this department we have to notice the revival of older methods of practice. Due prominence is now given to other indications besides supplying artificially the deficiency in iron. There is, in fact, a tendency toward discarding the ferruginous preparations. This is the result, I believe, of the use of very large doses of iron. Schultz and Strübung[49] found that some cases of chlorosis, if there was no gastric catarrh, were benefited by the use of sulphur. This treatment, recommended, I believe, by Graves, the editor found to be of special advantage in the anæmias of the Tropics, where constipation is a marked symptom. In the great majority of cases, however, the best results are obtained by giving iron at the same time. A prominent fact concerning anæmia and chlorosis, in his experience, is that they show a very decided tendency to spontaneous recovery, if not caused by serious organic lesion. Dr. Clark[35] bases his treatment of chlorosis upon his theory of fæcal toxæmia, and gives elaborate directions as to diet, hygienic measures, and the combination of laxatives with the iron. Prof. Nothnagel[50] recommends change of air, short baths, and the use of rather small doses of iron. Dr. André[44] pays special attention to restoring the functions of the liver by the employment of hydropathy, electricity and hepatic stimulants. Dr. Hutton[51] believes that the majority of cases of chlorosis require local uterine treatment. Some interesting observations have been made by Ch. Lejard to establish the effect of sea-baths upon the amount of hæmoglobin in the blood, the activity of its reduction in the tissues, and upon the pulse and respiration. The functions named are generally stimulated, especially at regular intervals, after each series of about seven baths. The sesqui-bromide of iron in jv–v gr. doses, in *dragées*, is recommended by Dr. Hecket.[52] Dr. Maragliano[53] recommends the following formula:—

℞	Emuls. amyg. amar., ...	℥x.
	Pulpæ splenicæ, ...	℥jv.
	Spt. vini gallici, ...	℥ij.

To be given in the 24 hours.

TRANSFUSION OF BLOOD.—INTRAVENOUS AND INTERSTITIAL INJECTIONS.—ACUTE ANÆMIA.

The following plans of medicinal treatment for the arrest of hæmorrhage have been advocated. *Les Nouveaux Remêdes*[54] recommends the nettle-juice, of which a permanent syrup has been prepared by Paneau. L. H. Petit[55] reports several cases of rebellious hæmorrhage controlled by the application of revulsives to the liver. Finally, hypodermics of atropine[56] have been given successfully in severe hemoptysis.

Before entering into the subject of tranfusion and other allied therapeutic means, it is well to notice the observations of Mosso,[1] Marigliano,[2] Silbermann,[16] and others, who show how easy it is to produce disintegration of the red corpuscles, and that saline solutions and alien blood have this property. It will be also instructive to introduce this subject with the observations of Hunter[57] on the duration of the red blood corpuscles after transfusion. "We have seen," says Hunter, "how, in the dog, in conditions of health, an excess of corpuscles, amounting to as much as 20 to 30 per cent., is got rid of in the course of a few days, the rate of destruction being as great as 5 or 6 per cent. or even more daily;

and we have further had reason to believe that man will approach very closely to the dog in respect of the activity of his various bodily functions. In man, however, we can rarely transfuse a quantity of blood equivalent to more than 5 or 6 per cent.,—a quantity probably utterly insufficient to influence in any material way the general mass of blood, since its destruction under ordinary circumstances may be expected to occur within a day or two at most. But if it be remembered that in pernicious anæmia we are transfusing these corpuscles into an organism whose own blood corpuscles are being destroyed with undue rapidity, it is doubtful whether the introduction of corpuscles under such circumstances can be of the slightest value, if the value of the operation is to depend on their continuance in life for some time afterward." The increased supply of oxygen might explain the beneficial effects of transfusion, but of course, this depends entirely on the duration of the corpuscles. Dr. Hunter is forced to conclude that the hæmoglobin introduced is the important factor in transfusion. It is probably utilized by the spleen and bone marrow.

Much attention has been paid to certain substitutes for the operation of transfusion. A very interesting paper by Dr. Pregaldino,[58] of Ghent, presents the arguments against intravenous transfusion or injections, and supports by numerous experiments the advantages of the subcutaneous injection of saline solutions. He quotes the opinions of Kronecker, Sander, Eulenberg, Landois, to the effect that the cause of death in acute anæmia is not so much the diminution of the number of red corpuscles, as it is the ischæmia of the respiratory and circulatory centres which results from the lowering of the blood pressure. A proof of this is found in the fact that a standard of 500,000 red blood corpuscles to the cubic millimetre is compatible with life in chronic anæmia, whilst in fatal acute anæmia the standard seldom falls below 2,000,000. In weighing this argument it should be remembered that the requirements of blood supply must be quite different in an anæmic from what they are in a healthy subject.

The strongest argument in favor of Dr. Pregaldino's method is its harmlessness. He has found by experiment that it is invariably successful if the animals have not lost more than one-half the volume of blood. They may recover even after losses of two-thirds of the volume, provided the bleeding has not been very rapid.

The solution employed consists of 6 grams of crystallized common salt to 1 litre of boiled water. The liquid should be filtered and boiled before using. The author employs, with antiseptic precautions, syringes of the capacity of 20 or 25 cubic centimetres, and he has injected as much as 60 cubic centimetres in one locality. The total amount injected should be about one-half of the volume of blood lost. The absorption is favored by massage, and by keeping the fluid at the temperature of the body. Klopsteck[59] and Hegar[60] present similar views to those of Pregaldino.

C. E. Jennings of the Cancer Hospital, London, reports three successful cases of intravenous injection of saline solution. We have also a favorable report from M. Kahan.[61] Lichtheim[62] reports five cases of chronic anæmia, in four of which the operation was followed by immediate bad effects.

Von Ziemssen[63] proposes the use of subcutaneous injections of defibrinated blood. The strictest antiseptic precautions are enjoined. 300 grams of blood are obtained from the donor and are kept at a temperature between 37° and 40° C. An anæsthetic is given and the injections are made in the thighs, massage being performed at the same time by an assistant. The hypodermic syringe used holds over 15 grams, and the author has given as many as fourteen injections at one sitting. An ice-bag is applied over the seat of the punctures. The operation has never been followed by fever, albuminuria or hæmoglobinuria. The suppuration which occurred in two cases was due to avoidable accidents. The amount of hæmoglobin in the blood may be doubled in the first twenty-four hours after the operation, and though it subsequently decreases, it remains at a higher standard than before. By this treatment Dr. Ziemssen has cured severe cases of anæmia in a month's time. It remains to be seen whether the operation is serviceable in pernicious forms of the disease.

Four cases of transfusion are reported by Dr. E. E. Allen.[64] Two of them were successful. The method of Annandale was employed in a case of Dr. Warrington Howard.[65] Six oz. of blood were received in a vessel containing 2 oz. of a solution of sodium phosphate of the strength of 1 in 20. The mixture was kept warm and was constantly stirred. A glass syringe of 3 oz. capacity was used. Mr. Allen[66] exhibited a new form of apparatus for transfusion before the Detroit Academy of Medicine. Dr. Valera[67] reports one case of chlorosis successfully treated by rectal injections of defibrinated blood. Dr. Escorileada[67] found good results from the use of a solution of blood and ammonium salt through the atomizer.

PERNICIOUS ANÆMIA.

Pathogenesis.—The observations of Reyher, of Dorpat, as to the parasitic nature of pernicious anæmia appears to be confirmed by J. W. Runeberg,[68] who describes 19 cases from the Helsingfors hospital, 12 of which were infected with the bothriocephalus latus, and 1 with lumbricoid worms. Only 1 of these cases terminated fatally,—a result to be contrasted with the previous record of 9 fatal cases. Runeberg believes the anæmia to be caused by disturbances of the intestinal function, and by reflex irritation, even when the parasite is the anchylostomum duodenalis. Lichtheim,[69] of Berne, reported 11 cases to the Sixth German Congress for Internal Pathology. Two of these were affected with the bothriocephalus latus, but they did not differ in their symptomatology from the others, and one of them was not cured by the removal of the parasite. In the discussion of the above paper von Jürgensen reported an acute case of pernicious anæmia, permanently cured after the administration of male fern, and the passage of large masses of bacterium termo. Dr. Litten, of Berlin, in the same discussion, reports 14 autopsies that do not favor the parasitic theory of the disease. Dr. Paul Fabre,[70] in his practice among miners, has never met with the anchylostomum, nor does he believe that miners are specially subject to anæmia. The causes of anæmia amongst them are the ordinary ones of traumatic hæmorrhage, overexertion, alcoholism, helminthiasis, lead poisoning, malaria, etc.

The editor cannot recall a single instance in which pernicious anæmia has developed as a consequence of intestinal parasites. But when a tape-worm is allowed to remain for a long time in the intestines, he has observed, especially in women, after the cure a more or less permanent condition of anæmia, lithæmia, dilatation of the stomach, and hypochondriasis.

F. E. Georgi, of Berlin,[71] publishes a case of pernicious anæmia with primary hepatic symptoms. Numerous gall-stones were found in the gall-bladder. The author believes that in this case, and in nine others reported, the persistent presence of the bile acids in the blood gives rise to a condition of hæmoglobinæmia,

TABLE OF CASES OF PERNICIOUS ANÆMIA.

AUTHOR AND REFERENCE.	SEX.	AGE.	CONDITION.	PREVIOUS HISTORY.	APPEARANCE.	RETINAL HÆMORRHAGES.	DIARRHŒA.	VOMITING.	FEVER.	OTHER SYMPTOMS.
Copeman[75]	F	27	Single	Rheumatic	Jaundice. No emaciation.	Yes.	No.	Yes.	Yes.	Epistaxis. Pain in the long bones. Systolic murmurs.
Copeman[75]	M	32	Leather finisher.	Rheumatic	Very anæmic. Yellowish tint.	No.	No.	No.	Yes.	Epistaxis. Paroxysmal cough. Systolic murmurs. Albuminuria.
Copeman[75]	M	57	Brewer's man.	Rheumatic	Emaciated. Pale. Aged looking.	Yes.	Yes.	Yes.	Yes.	Dry cough. Pain in long bones.
Copeman[75]	F	33	Married	Pregnancy	Pale brownish yellow. No emaciation.	Yes.	No.	Yes.	Yes.	Œdema of legs. Pain in long bones.
Copeman[75]	M	47	Policeman.	Malaria.	Very anæmic. No emaciation.	Not examined	Yes.	Yes.	Yes.	Profuse sweating. Pain in long bones. Murmurs.
Nothnagel[80]	....	....	....	....	....	....	....	....	....	Diminution of the alkalinity of the blood. Rapid development of leukæmia at the approach of death.
W. Tyrrell Brooks[81]	F	38	Married	Rectal hæmorrhages.	Pale. Not emaciated.	Yes.	No.	Yes.	Yes.	Sternal pain. Subcutaneous hæmorrhages. Apparent recovery, followed one year later by relapse and death.
Cole[82]	M	35	Cabman	Rheumatic	Pallor	Yes.	....	....	Yes.	Paroxysmal cough. Murmurs. Apparent recovery.
Bates[77]	....	49	....	....	Jaundice	....	Yes.	Yes.	....	Fatty liver. Atrophy of stomach.
Asta-Buraga[83]	M	40	Baker	Alcoholic	Emaciation. Jaundice.	....	No.	Yes.	Yes.	Constant pain at epigastrium.
Asta-Buraga[83]	M	52	Engineer	....	Jaundice. No emaciation.	....	No.	Yes.	Yes.	Epigastric pain. Pneumonia.
Asta-Buraga[83]	M	50	Butcher	....	Pallor. No emaciation.	Yes.	Yes.	Yes.	Yes.	Transient albuminuria and casts.
Asta-Buraga[83]	M	36	....	....	Pallor. No emaciation.	Yes.	No.	No.	Yes.	Epistaxis. Murmurs.
Asta-Buraga[83]	M	39	Cabinet-maker.	....	Pallor. Emaciation.	Yes.	No.	No.	No.	Improvement. Disappearance of retinal hæmorrhages.

and that the bone-marrow takes on compensatory activity to make up for the destruction of red corpuscles. Dr. Lecaze[72] reports a case of intense anæmia due to hypertrophy of the liver. Reference to jaundice as a symptom will be found in a considerable proportion of the cases reported. Dr. E. A. Ewal,[73] however, after reporting a case of short duration in which no primary lesion of the liver was found, criticises the views of Georgi. The jaundice in his opinion is due to the rapid destruction of red corpuscles. This furnishes the liver with an excess of hæmoglobin, out of which the biliary coloring matter is formed. The excess of the latter, taken up by the circulation, causes the jaundice.

Mr. Jessop[74] reports three cases of pernicious anæmia in persons living in houses much exposed to sewer gases.

Morbid Anatomy.—The changes in the blood have been investigated by S. M. Copeman,[75] who confirms the observation of Eichhorst as to the existence of small, spherical, highly colored corpuscles. He observes that the coloring matter is not necessarily dissolved in the plasma, but may remain in amorphous masses. The colorless protoplasmic granules he found to be more numerous than in health. On rapidly drying the corpuscles on a slide they break down readily, forming crystals of hæmoglobin. Where the corpuscles do not break down, we find amongst them brown masses of pigment, probably hæmatin.

As a result of the study of 250 cases, Ch. Geehinnyden[76] concludes that the bone marrow is always affected in this disease. Two cases are reported by Bates,[77] and two by F. P. Kinnicutt,[78] in which the mucous membrane of the stomach was found atrophied. Delafield[78] believes that these changes must be secondary, because we do not find in starvation the symptoms of pernicious anæmia.

Nervous symptoms and lesions are reported in the course of this disease by Lichthein.[69] He has observed three cases in which the symptoms of tabes dorsalis were secondary to the anæmia. In two of these, examined post-mortem, the lesions of sclerosis were found. R. Lépine[79] reports a case of extensive atrophic paralysis developing in the course of pernicious anæmia.

Treatment.—The curable cases seem to have been favorably influenced by arsenic: Asta-Buraga,[83] Cole,[82] Allbutt.[84] One case of Lépine's appears to have been benefited by an intravenous injection of 400 cubic centimetres of saline solution. See also the effects of anthelmintics reported under pathogenesis. The chapter on transfusion should also be consulted.

HODGKIN'S DISEASE.

Cases are reported by C. P. Putman,[85] Harold Williams,[86] James Macready,[87] F. Treves,[88] Forschheimer.[89] The majority of successful cases were benefited by the use of arsenic in gradually increasing doses. In the case of Macready, electrolysis was ineffectually tried. All the cases were males. The cases of Putman and Williams ran a rapid course.

Hodgkin's disease is perhaps more common in the Southern States than in the North, whilst the reverse is true of pernicious anæmia and lukæmia. The editor has to report the case of a child 5 years old, of Key West. All the glands accessible to physical exploration were affected. The duration of the disease was one month, from the first enlargement of the cervical glands to the fatal termination. During the editor's residence of eight years in the South, he has met with six cases of Hodgkin's disease, one of pernicious anæmia, and none of leucocythæmia.

LEUKÆMIA.

Pathogenesis—C. W. Earle[90] reports the case of a leukæmic woman who became pregnant, and was delivered of a non-leukæmic child.

Morbid Anatomy.—Dr. Prus[91] found crystals of leucin in the blood only in lymphatic leukæmia. The number of blood-plates, and the activity of the leucocytes he found to be increased.

Symptomatology.—Von Bamberger,[92] of Vienna, calls attention to the inspiratory dyspnœa met with in leukæmia, which is not in proportion to the oligocythæmia, the lukæmia or the oligochromæmia. He believes it may be caused by excitation of the respiratory centres by matters retained in the blood.

Cases.—Cases are reported by J. M. Ball[93] and H. Culberton.[94] One case of very rapid course is reported.[95] The patient was ailing only ten days. From the post-mortem appearances, however, it may be judged that the spleen must have been diseased for some time previously. Wilh. Retslag[96] reports a case of about six months' duration in a girl six years old. She had also symptoms of pernicious anæmia. One case is reported by W. Collier;[97] four by Flinterman,[98] of Detroit, and one by Stewart.[99] Of these 10 cases, 7 were males, and only 2 were of the lymphatic form. Two cases[95,100] are reported in which the retinal symptoms were the first to present themselves.

Treatment.—The treatment by inhalations of oxygen, suggested by Kirnberger, has been tried by Sticker[101] and Pletzer.[102] The quantity used was from 30 to 90 litres a day. In all the cases the treatment was followed by marked improvement of all the symptoms, except the enlargement of the spleen. But the improvement was not permanent. It may be said, however, that the treatment was not given a thorough trial. Prof. Potain[100] advocates the use of quinine and arsenic, with the employment of hydrotherapeutics. The case of Collier[97] shows the temporary improvement derived from the use of arsenic. The following formula of Mosler is given by Henry:[103]—

℞	Ol. eucalypti, ...	gtt. 100.
	Piperini, ...	
	Ceræ albæ, ...	aa ʒj.
	Pulv. altheæ, ...	ʒij.

M. et. ft. pil. No. C.
S.—Three to five pills thrice daily.

Mosler recommends also the use of large doses of quinine and of the faradic current.

A systematic study of the different forms of anæmia will be found in a series of papers published[103] by Dr. Henry, of Philadelphia.

THROMBOSIS.

The German pathologists have been engaged in a discussion concerning the formation of the white thrombus. Eberth and Schimmelbusch[13] maintain that the white thrombus is the result of a peculiar viscous metamorphosis and aggregation of the blood plates. This they term conglutination, which is to be distinguished from the separation of fibrin or coagulation. Hanaw and Weigert[13] impugn the correctness of these conclusions, and the latter more particularly insists upon the important part that the white blood cells play in the thrombotic process, as was shown by Zahn. Fibrillar fibrin is also present, though it may require special staining. Eberth and Schimmelbusch contend that their description covers the initial steps in the process,—the presence of leucocytes and fibrin being secondary manifestations. Löwit agrees with this, except that he does not consider the blood plates as normal constituents of the blood, but as the result of separation of the globulin.’ Compare Mosso,[1] Osler[14] and a résumé of the subject by Prof. Ribbert.[15]

HÆMOGLOBINÆMIA.

Silbermann[16] makes a valuable experimental contribution to our knowledge of this condition. The experiments were made upon frogs, rabbits and dogs, by injection of substances which are known to cause a solution of hæmoglobin. He generally used lake-colored blood. The result was invariably a vast accumulation of blood in the venous system, and the deposition of layers of leucocytes along the walls of the vessels, forming at times obstructive plugs. The symptoms produced under these circumstances were: dyspnœa, cyanosis, heightened reflex irritability, high temperature and frequent pulse, convulsions, disturbances of equilibrium, hæmoglobinuria, nystagmus, pendulum motions of the head, reactionless pupils, and frequently general clonic spasms preceding death. All these symptoms are due to arterial anæmia. The stagnation of blood in the veins is caused by the extraction of the fibrin-ferment from the white blood cells, venous blood containing more ferment than the arterial.

SCURVY AND PURPURA.

Purpura has been divided[17] into three forms: the aqueous, the saline, and the vascular. In the first the specific gravity of the blood may be reduced to 1035. In the saline form the fibrin is held in undue solution by the alkalinity of the blood. This includes the scorbutic cases. In the vascular form there is either a structural or paralytic disturbance of the vascular walls. Two epidemics of scurvy are recorded by J. J. Molleson,[18] of Perm, caused by bad crops of corn and potatoes. The disease attacked over 7000 persons.

Treatment.—The following mixture is recommended in purpura:—

℞	Syrup of iron superphosphate, ...	℥jss.
	Solution of hydrogen peroxid (10 vols), . .	℥jss.
	Pure glycerine, ...	℥jss.
	Distilled water to make	℥vj.

Tablespoonful three times daily.

Hydrochloric acid is recommended in the saline variety of purpura, and fresh air and turpentine in the vascular. The London *Lancet*, commenting on the death from scurvy of a sailor from Calcutta, observes: "Lime juice no doubt is an excellent preventive; but if ships were provisioned in accordance with the opportunities now afforded by cold storage and hermetically sealed tins, it would become unnecessary." Alex. Faulkner[19] has employed salicylic acid with success in the treatment of scurvy at Peshin, S. Afghanistan.

HÆMOPHILIA.

Pathogenesis.—H. Hughes[20] believes that this disease is caused by lesion of the vaso-motor centre, and consequent vascular paralysis. This theory has been accepted by Dr. Oliver, of London. Dr. Hughes rejects the theory of changes in the blood and the vessels because no such changes have been demonstrated. But he fails to demonstrate the lesions that his hypothesis would have us to localize in the vaso-motor centres. Furthermore, we do not find any evidences of vaso-motor palsy in hæmophilia, beyond the signs of deficient arterial tonus that is found in all anæmias. Finally, it may be observed that the spontaneous arrest of ordinary hæmorrhages is brought about by this very condition of vascular relaxation, which is supposed by Dr. Hughes to be the only cause of persistent hæmorrhage. Vascular relaxation means retardation of the blood current, which is one of the essential factors of blood clotting. It is evident that no explanation of these hæmorrhages can be satisfactory that does not account for the deficient formation of occluding thrombi. That the blood of these patients when removed from the body does coagulate, signifies nothing. The tissues and the products of their metabolism are probably the source of hindrance. We have an example of this in the non-coagulability of the blood sucked by the leech. The theory of Dr. Hughes is particularly defective in its application to malarial hæmaturia. This is frequently not a hæmorrhage at all, and is always a manifestation of profound blood changes.

Cases.—One reported by Savoye.[21] A girl, 8 years old, with good family history, excepting the grandmother, who died of cancer. Dr. Blake[22] reports a boy 5 years old with a family history of phthisis. Dr. Chambers[23] reports four cases: (1) Male, aged 30 years, with rheumatic family history. Two other members of the family are bleeders. (2) A boy 6 years old. No family tendency. (3) A boy 3 years old, without any family tendency. Hæmorrhages coming on after diphtheria treated with large doses of bichloride of mercury and iodide of potassium. (4) Male, 48 years old, without any family tendency. James Graham[24] reports a case of a male, age 20 years. Two brothers are bleeders and five sisters are not. No previous family tendency. Herbert W. Page[25] gives the history of a male, 22 years old. The maternal grandmother was a bleeder. The following or second generation consisted of 11 children, 6 males, who died of unknown causes, and 1 male and 4 females, who are all bleeders. One of the latter is the mother of the patient. Her children alone of the third generation are bleeders. She has 4 daughters and 3 sons. Of these 3 daughters and 1 son are bleeders. The fourth generation consists of 2 children of the eldest sister of the patient, who is a bleeder. These children are not affected. Another case of a male, 52 years old, is reported.[26] There is no previous family tendency. Of his 7 brothers and sisters, 1 brother and 1 sister's son are affected. Henry Skelton[27] reports three cases, all males, aged respectively 12 years, 16 months, and 17 months,—the latter with a history of asthma on the mother's side. Edmund C. Wendt[28] reports a very interesting case, with autopsy of a newborn male child. Parenchymatous hæmorrhages probably commenced *in utero.* The parents of the child were healthy. Another case of a newborn child is reported by J. K. Caldwell.[29]

DISEASES OF THE SPLEEN.

Regeneration of the Spleen.—An experimental removal of the spleen of the fox was followed by the formations of a splenic nodule in the splenic region, and also numerous new formations in Peyer's glands, in the adipose tissue of the mesentery, and in the liver.

Movable Spleen.—A case in an epileptic woman is reported by A. Lükin.[101]

Abscess of the Spleen.—Mr. Choudhoory,[105] of India, reports that of 30,000 cases of malaria treated, only three presented abscess of the spleen. He describes one, of slow development. The abscess was freely opened, after pointing, and was treated successfully by drainage, boracic lint, and iodoform.

Hypertrophy of the Spleen.—Under the name of splenic pseudo-leukæmia, we find the careful record of a case by M. Potain.[106] It was one of emphysema, with progressive pallor, emaciation, weakness, and epistaxis for four years. There was no fever, no leukæmia. The red corpuscles were reduced to 2 millions per millimetre, the hæmoglobin to one-third of the normal, and the spleen was very much enlarged. There was albuminuria. No history of syphilis or malaria. Directions are given in this paper for the physical examination of the spleen. A similar case, in its last stages, ten days before death, is described by R. G. Shore.[107] This patient had pus and blood in the urine. The temperature was febrile, and death occurred from exhaustion. The spleen, weighing 5½ lbs, is described as normal in appearence. It is probable that the acute congestion of the organ obscured the sclerotic changes that are found in these cases. In the lymphatic form of pseudo-leukæmia there is—as represented in the plate accompanying an article of Dr. Murchison's in the London Pathological Society Transactions—a hypertrophy of the Malpighian bodies which gives rise to a characteristic variegated appearance.

The Ague Cake.—Fazio[108] has obtained in two cases a decided reduction of the tumor after thirty-two and forty-five injections of quinine. The dose was of gr. iij–jv in ♏xv of distilled water. An ice-bag was applied after the operation, and all pain and febrile reaction was thus avoided. V. Richards[109] has employed with success, as recommended by natives of India, the administration of borax in lumps of from 30 to 40 grains, to be dissolved in the mouth, mornings and evenings.

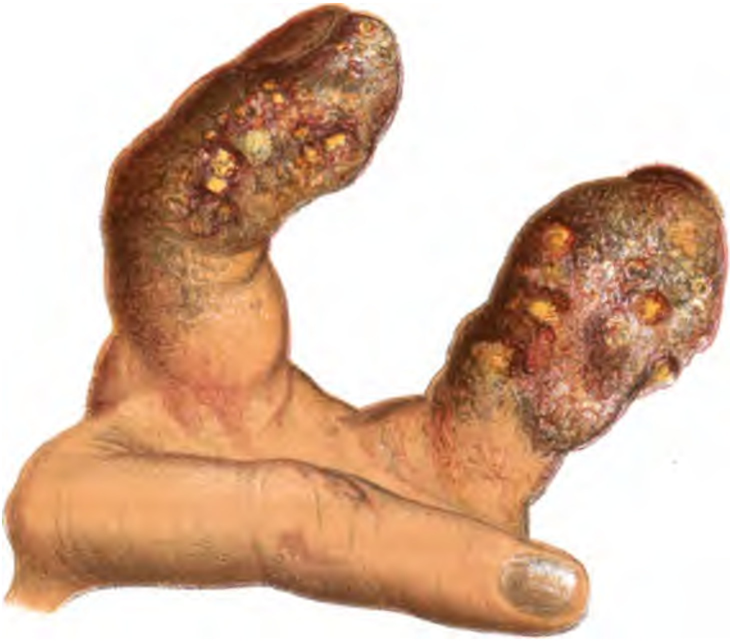
One case of *hydatid* cyst is reported by Besançon.[110] The tumor was indolent. The patient died of pneumonia.

TUBERCULOSIS.

Pathogenesis.—The State Board of Health of Maine has published an interesting report, by A. G. Young, summarizing the information concerning bovine tuberculosis, and its relations to man. Experiments were made by V. Galtier[111] with tuberculous masses which were subjected to heat, desiccation, water, freezing, putrefaction, and the action of salt. He proves that the different processes for the preparation of meats do not insure absolute safety from tuberculous infection. W. Sibley[112] read before the London Pathological Society a report on avian tuberculosis as it occurred on a farm in Surrey. The disease generally appeared at the age of two years. Caseous masses were found in the intestinal walls. The cervical glands, larynx, trachea and vertebræ were also affected. A bacillus was found apparently identical with the human. A hereditary predisposition was shown.

With respect to the hereditary transmission of tuberculosis, it is difficult not to agree with Firket,[113] who doubts that prenatal tuberculosis can have its source in tuberculous semen: if such were the case the development of the embryo should be affected and a monstrosity should result. This conclusion is not invalidated by the positive proof, advanced by Landouzy and Martin[114] that the semen of tuberculous animals does contain the tubercle bacillus. Six guinea-pigs out of sixteen were successfully inoculated with semen of tuberculous rabbits. This problem is far from being solved. It is important to remember in this connection that the tubercle bacillus has been found in the placenta. In another contribution,[115] Landouzy calls attention to the frequency of tuberculosis in early life. During the first quarter of 1887, seven cases were observed at Tenon Hospital, aged respectively six weeks, and three, six, seven, ten and twelve months.

Evidences of inoculation of tubercle in the human subject are multiplying. We have for instance the cases of tuberculous infection in the site of circumcision, reported by Bergmann, Hofmokl,[116] Elsenberg,[117] Lehmann.[118] We have the probable inoculation of the stump of the forearm of a child by a nurse who was affected with lupus, reported by Wahl.[119] We have also a most conclusive observation by E. Leser.[120] The patient, a healthy woman, had a small wound of the last phalanx of the thumb. The wound refused to heal, and became painful and ulcerated. In about a year a similar ulcer appeared on the opposing surface of the index finger. In about two years' time there appeared, first a suppurating lymph gland of the inside of the arm, and a large cold abscess under the pectoral muscle, with slight involvement of the third rib. The lymph gland was not examined, but the other lesions were found by histo-bacteriological investigation to be tubercular. F. Treves[121] also reports a case of probable inoculation of a lacerated wound of the great toe. The tubercular process



Digital Tuberculosis.

[illegible]

followed a course similar to that of chronic pyæmia, extending over a period of five years and causing superficial abscesses, and involvement of the vertebræ, the knee, the lymphatic glands, the testicles, the prostate, the kidneys, the lungs, the peritoneum.

It must be admitted that the direct inoculation is not always conclusively proven. This is particularly true of genital tuberculosis. It has been suggested[22] that the observations of Verchère and Fernet are not conclusive on this point, because we find that the tubercular lesions are not apt to occur in the parts most exposed to the contagion, and further, that genital tuberculosis is not rare amongst children who are not exposed by venereal contact. The subject is further complicated by the possibility that the bacillus may be introduced without producing any local effect at the point of entrance.[120]

Verneuil[123] shows the rarity of the coexistence in the same individual of tuberculosis and lithiasis. An experimental contribution is made by Daremberg[114] to show the virulence of the fresh spinal marrow of tuberculous animals not affected with meningeal tuberculosis. He also succeeded in producing tubercular meningitis, by introducing tubercular matter under the cranium, by trephining.

Morbid Anatomy.—Cornil[124] describes the histogenesis of tubercle as follows: "Under the influence of the bacillus the phenomena of kariokynesis are stimulated in the connective tissue cells, and in the endothelial and epithelial cells, thus producing the epithelioid cells. The bacilli wander into the capillaries where they become fixed, causing a wandering of white cells which collect around the tuberculous new formation. The formation of tubercular thrombosis in the spleen and kidneys is described by Dietrich Nasse.[124] Brissaud and Toupet,[114] in their studies of hepatic tuberculosis, find that the tubercles develop primarily in the capsule of Glisson. They hold that there is a specific tuberculous cirrhosis of the liver of a chronic inflammatory nature, without a tuberculous new formation proper. They report considerable difficulty in staining the bacillus in the liver. Dr. Barlow[125] contributes a case of tubercular ulcer of the stomach in a child with general tuberculosis. It was situated in the greater curvature near the cardiac orifice.

Symptomatology. — The attention of observers has been concentrated toward the local forms of the disease. These will require a separate chapter. Several cases will be mentioned here to illustrate peculiarities in the course of the disease. An interesting case is reported by Favel.[126] A young man recovered, after change of climate, from pulmonary consumption. He suffered subsequently for many years with glandular tuberculosis. At the age of 51 he had the shoulder resected for tuberculous disease, and appears to have recovered completely. Delafield[127] reports a case of acute tuberculosis of the lymphatic glands. The autopsy indicates that a slow and latent tubercular process had been going on for some time in the mesenteric glands. A case of latent tubercular meningitis is reported by Dr. Belfield,[128] with sudden symptoms of narcotic poisoning.

Treatment.—The use of guaiacol internally and by inhalations is recommended by Sahli and Max Schüller.[129] Dujardin-Beaumetz[130] has been experimenting with inhalations of sulphurous acid. The method consists in burning sulphur, 300 grains to the cubic yard, in a close room. Twelve hours later the patient enters the room, and is allowed to remain eight hours. In some cases the bacillus disappeared from the sputa and the symptoms were relieved, whilst in others the suffering was increased. J. V. Froschauer,[131] in discussing the treatment of local tuberculosis by the acid phosphate of lime, remarks upon the alkalinity of the tissues of herbivorous animals. These show a marked predisposition to tuberculosis. The comparative resistance of carnivorous animals to infectious diseases may be due to the production of acid calcium phosphate from the decomposition of nitrogenous food. Lutton,[132] of Rheims, recommends the phosphate of copper in the nascent state, in the following formulæ:—

℞	Neutral acetate of copper, ...	1 centig.
	Phosphate of sodium, crystal, ...	5 centig.
	Liquorice and glycerine enough to make one pill.	
℞	Neutral acetate of copper, ...	5 centig.
	Phosphate of sodium, ...	50 centig.
	Mucilage, ...	125 grams.
	Tablespoonful doses.	

For hypodermic medication 1 part of the recently precipitated phosphate of copper is dissolved in 5 of glycerine and water. This treatment is followed by the use of a tonic containing phosphate of sodium.

Petit and Bergeon[114] contribute papers in reference to gaseous enemata. The reports[133] from America concerning this procedure, by Bruen, F. C. Shattuck, and Pepper, are not as favorable as the earlier publications seemed to promise.

Concerning the *attenuation of the tubercular virus*, Gosselin reports utter failure with the following methods: passing the virus through comparatively refractory animals, obtaining it from the milder local manifestations of the disease, and, finally, the previous preparation of the system by daily injections of iodoform, and the bichloride and iodide of mercury. Iodoform was found to have some effect toward checking temporarily the disease when it was already present. The same effect was determined for tannin by Raymond and Arthand.[114] These observers were unable to produce artificially the zooglœa formations that they consider to be attenuated forms of the bacillus. Thorkild Rovsing[134] has proven by inoculations of the anterior chamber of the eye of rabbits with tuberculous matter thoroughly mixed with iodoform, that this drug has neither a germicidal nor an antiseptic action upon the tubercle bacillus. Florand[135] has shown that the exposure for one month in an atmosphere of oxygen or of mercurial vapor destroys the virulence of tubercle, but that dry air has no effect whatever. Parenchymatous injections of oil of turpentine are recommended by Riva[136] in pulmonary tuberculosis. He uses a solution of one part of turpentine in ten of water, adding water until the mixture becomes cloudy.

SCROFULA.

Pathogenesis.—M. Arloing[137] found as the result of his experiments that true glandular scrofula produces no lesion when inoculated into the rabbit; nor does the passage of the scrofulous virus through two generations of guinea-pigs in any manner intensify its virulence toward rabbits. On the other hand, the attenuated virus of tuberculosis, such as is found in its local manifestations, is intensified by passing through the body of the guinea-pig. This is strong evidence in favor of the view that scrofula is not an attenuated or modified form of tuberculosis. But F. Eve[138] objects to these experiments that the original scrofulous material was all obtained from one subject. Eve chose his material from ten cases taken at hazard. The inoculations produced invariably tuberculosis in the guinea-pig, and four times in the rabbit. If the virus was first passed through the guinea-pig, then it was invariably virulent to the rabbit. Eve could find no essential anatomical differences between the lesions produced by the strumous and the tuberculous inoculation. He found the bacilli in small numbers in the scrofulous glands, but did not find that they assumed the forms described by Malassez and Vignal under the name of *tuberculose zoogléique.* The fact that scrofulous glands contain tubercle bacillus, does not prove that the scrofulous gland is primarily a tuberculous gland. The view taken of this question by Noeldechen[139] is that scrofula is the most favorable soil for the development of the tubercle bacillus. Rable[140] suggests that we may perhaps find, in the discovery by Freund, of cellulose in the blood and tissues of scrofulous patients, a distinctive feature between the two affections. He further mentions the tendency of scrofula to symmetrical manifestations, and the fact that the bacillus is not constantly found in scrofulous glands. Rable tabulates 1000 cases of scrofula as follows: 79 had scrofulous parents, 446 had tuberculous parents, 356 lived in damp dwellings, 25 were subjected to other bad hygienic surroundings, 69 could be ascribed to acute infectious diseases, 14 to vaccination, 7 to decrepitude and 4 to consanguinity of the parents.

Treatment.—In the paper by Noeldechen already referred to will be found an account of the treatment of scrofula at Kösen by the brine baths. Van Merris[141] has published a work on scrofula and sea-bathing. A. F. Suchard[142] advocates the use of thermal chlorine and sulphur baths, and sea-bathing. The salts are not absorbed, but they act by stimulating the peripheral nerves. Max Scheimpflug[143] furnishes information concerning the institutions for the treatment of scrofula in Europe. William Alexander[144] reports the advantages that have been gained by the establishment of a country house for scrofulous patients in connection with the Liverpool Workhouse Hospital.

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[Undated references apply to journals published in 1887, and original articles can be found by consulting the indexes of the respective publications.]

RHEUMATISM AND GOUT.

By N. S. DAVIS, M.D., LL.D.,

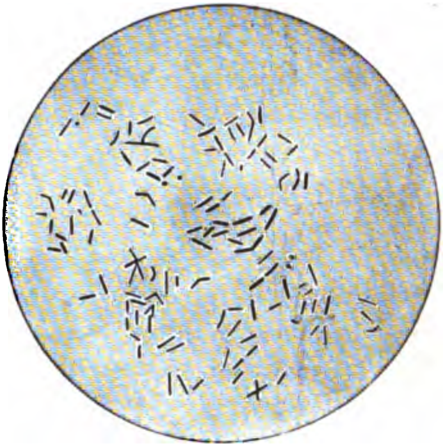
CHICAGO.

RHEUMATISM, ACUTE AND CHRONIC.

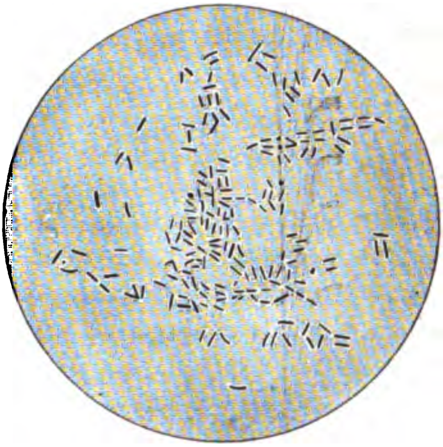
Etiology.—The causes that favor the development of rheumatic inflammation in its various grades of intensity, have attracted but little attention from the profession generally during the year 1887. A large number of articles have appeared in the medical press of Europe and America, concerning the therapeutic management of the various grades of rheumatism; but in only a small proportion of them is there any allusion to the causes that may have been efficient in producing the disease.

In the *Pacific Medical and Surgical Journal*, San Francisco, Cal., for February 15th, 1887, we find a leading article under the title of "The Chemical Nature of Gout and Rheumatism," the writer of which commences with the assumption that both these forms of disease depend upon the presence in the system of uric acid. Without presenting any facts in support of this assumption so far as regards rheumatism, he proceeds with an interesting theoretical explanation of the successive chemical changes that the nitrogenous elements of food undergo in passing through the digestive and assimilative organs, and arrives at the conclusion that "uric acid is one of the by-products" resulting from these complex processes. When the uric acid is developed, it is converted by the action of oxygen into urea and readily eliminated; but when the amount of oxygen present is deficient, the uric acid accumulates in the blood and may become the efficient cause of rheumatic or gouty inflammations, according to the previous predispositions of the individual. This theory of deficient oxygen to complete the metamorphosis of uric acid into urea as a cause of rheumatism can find no support from the habits and surroundings of the great majority of rheumatic patients who are found among the manual laboring class, both male and female, and who, by their occupations, are forced to the daily use of a maximum quantity of atmospheric oxygen.

Dr. Alfred Mantle,[1] of Stanley, England, details the results of his investigations regarding the presence of bacteria as an etiological factor in rheumatism. The extent and method of his investigations are indicated by the following quotations: "A typical case of acute rheumatism being under his care, under strict antiseptic precautions, and by means of a sterilized hypodermic syringe, about a drachm of serum was withdrawn from the knee-joint. Several sterilized tubes of gelatinized meat-infusion were at once inoculated, and in each tube a copious growth took place. In six other cases serum had been withdrawn in the same way, with like results. Serum being not always available, whilst blood was, and feeling it to be of equal importance to discover if organisms were present also in the blood, in sixteen cases of acute rheumatism, including some which would be called subacute, blood had been taken from the general circulation, with every precaution to prevent contamination from without, when in every case bacteria had been found." In addition, the blood of ten cases of decidedly chronic rheumatic affections had been examined by him for bacteria with the same results as in the acute form of the disease.

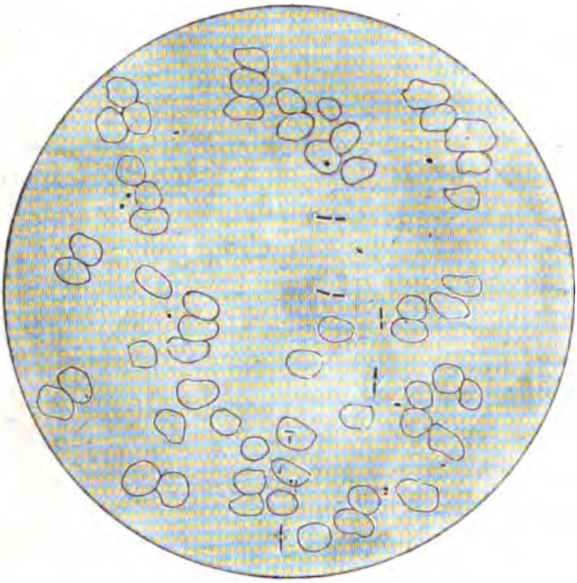


(*British Med. Jour.*)



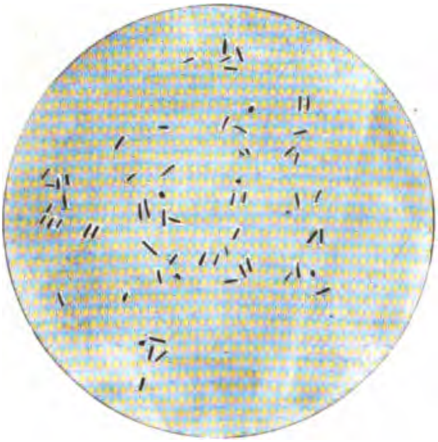
(*British Med. Jour.*)

The organisms discovered by Dr. Mantle are described as consisting of a micrococcus and a small bacillus,—the former much the most numerous. They were easily stained with methyl-violet, fuchsin, or by Gram's method, and are represented in the following cuts shown herewith, copied from those accompanying the paper.



(British Med. Jour.)

These results are interesting and important, as they establish the fact of the existence of both micrococci and bacilli in the blood and the serous effusions accompanying acute and chronic rheumatism. Before any inferences can be drawn in relation to their etiological influence, however, the same examinations must be made of the blood and the serous effusions in non-rheumatic grades of inflammation, to determine whether their presence is limited to rheumatism, or found equally abundant in connection with a variety of other diseases; and the usual careful inoculations with pure cultures must be made in animals known to be capable of suffering from attacks of rheumatism.



(British Med. Jour.)

While Dr. Mantle evidently regards rheumatism as dependent upon bacteria, he fully admits the necessity of such further investigations as just indicated, and adds the positive declaration that it is not the organisms which directly produce the disease, but the chemical products resulting from their action upon the tissues. He claims to have found the cultivation of bacteria found in rheumatism and some other diseases to produce lactic acid fermentation in sterilized milk, which leads him to ask whether this acid may not be the chief ptomaine of rheumatic disease.

In connection with this subject it is proper to refer to a case related by Guttmann[2] to the Medical Association of Berlin, of a boy aged fourteen and one-half years, as one of multiple acute articular rheumatism. Pain and swelling attacked successively the knee-joints. In four days the pericardium became involved, followed by copious effusion, and in a very few days the patient died. Some of the effused fluid was taken from the knee-joint and also from the pericardial sac, and from these Guttmann cultivated on gelatin an abundance of cocci, described as the staphylococcus areas. But as the post-mortem examination revealed not only purulent effusion in the synovial and pericardial sacs, but abscesses and purulent infiltrations in both kidneys, and small abscesses in the left pectoral muscles, it is not easy to see why the case should have been reported as one of rheumatism, instead of pyæmia. Certainly the finding of cocci in such a case cannot be legitimately construed as throwing any light on the etiology of true rheumatism.

Prof. Pel,[3] of Amsterdam, has suggested that acute rheumatism is dependent upon malaria, and, in his clinics, has furnished statistics showing some apparent relation between the prevalence of rheumatic fever and intermittent. But the well-known fact that rheumatism in all its forms prevails in seasons of the year when malaria is least active, and in districts of country where ague is not known to originate, is a sufficient answer to the suggestion of Professor Pel.

Pathology and Pathological Anatomy.—Very few contributions have been made to medical literature during the past year having any direct bearing upon the pathology or pathological changes in rheumatism.

In a paper by Pitres and Vaillard,[4] on "Peripheral Neuritis in Chronic Rheumatism," are given the results of their investigations concerning the conditions of the peripheral nerve in three cases, in addition to those described in a paper presented by the same authors to the Société de Biologie the previous year. In all the cases marked changes were traceable in many of the nerves of the extremities. Some diffuse sclerosis was found in the white columns of the spinal cord, and one, traces of posterior spinal meningitis.

There was not sufficient uniform correspondence between the nerve lesions and the joint affections in any of the cases to justify the inference that the latter were dependent upon the former. For instance, in some of the articulations most affected the nerves showed but slight traces of any morbid changes.

M. Renaut[5] relates a case of a patient who had an attack of acute articular rheumatism each year for several years. After each attack he lost his hair and the nails from his toes and fingers, but the latter were subsequently replaced. These peculiar trophic changes were not accompanied by any symptoms of disease in the spinal cord. Such trophic changes as have been pointed out by Pitres and Vaillard in the nerve structures, and by M. Renaut and many others in the hair, nails, muscular and other tissues, are referred to by some writers as evidence of the nervous pathology of rheumatism. Several cases of acute cerebral rheumatism accompanied by hyperpyrexia, reported during the last year, have been regarded as having the same pathological significance. A much more reasonable inference would be to regard all these cases involving changes in the nerve structures, whether acute or chronic, intra-cranial or peripheral, as resulting from the development of the usual rheumatic inflammation in the connective tissue and neuralemma, instead of its more common seat in the same class of tissues connected with the articulations and other organs of the body. The same grade of morbid action that so uniformly leads to sclerosis and other trophic changes in the ligaments, synovial membranes, fasciæ and muscles, if located in the neuralemma and sheaths of the nervous structures, would as certainly cause sclerosis or atrophy in the latter; or when acute and invading the great nervous centres it could hardly fail to develop the highest grade of temperature and excitement.

Treatment.—While a careful review of the medical literature of 1887 has discovered but little that could be regarded as actual additions to the knowledge of etiology and pathology of rheumatism, it has revealed the results of treatment in a large number of cases, and of some remedies comparatively new. The therapeutic value of salicin, salicylic acid and the salicylates in the treatment of the different grades of rheumatism had only very recently become generally recognized by the profession, when early in the past year attention was directed to three new drugs,—antipyrin, kairin and salol. The first two are derivations of chinoline, and the last, introduced by Nencki and Sahli, appears to be a combination of the carbolic and salicylic acids. The use of these remedies in the treatment of acute and sub-acute articular rheumatism was commenced almost simultaneously by many physicians on the Continent of Europe, more especially in Germany and France. Dr. John Elliott[6] gives an interesting abstract of a report of the treatment by Fränkel[7] of thirty-four cases of rheumatism with antipyrin. The cases were all males between the ages of fourteen and twenty-eight years; thirteen were mild or sub-acute and twenty-one severe or acute with high fever. Nine of the thirteen cases and four of the twenty-one yielded so promptly to the influence of one gram (grs. xv) doses of the antipyrin repeated every three hours, that no doubt could be entertained regarding the direct controlling influence of the remedy. In only one case of the thirty-four did the remedy induce vomiting, and in another it had to be abandoned on account of a marked idiosyncrasy of the patient; while in all the remaining cases improvement, more or less rapid, took place. Its most direct and manifest effects were speedy relief of pain and reduction of temperature when given as stated by Fränkel, which indicates its special adaptability for the treatment of the early stage of the more acute grade of rheumatic fever.

At the Annual Meeting of the American Medical Association, June 8th, 1887, a paper on "antipyrin in rheumatism, its value and mode of action," was read by Dr. N. S. Davis, Jr.,[8] in which the writer gave the result of his use of antipyrin in the treatment of acute and sub-acute rheumatism, both in hospital and private practice. In some of his cases he gave the remedy in one gram (grs. xv) doses every three or four hours, and in other cases he gave thirteen decigrams (grs. xx) every six hours, making from five to six grams in the twenty-four hours. The results obtained by him were strictly analogous to and equally favorable with those reported by Fränkel, namely, the rapid reduction of temperature, prompt relief from pain, and general perspiration. For an explanation of the *modus operandi* of the drug in developing these therapeutic results, he refers to the experimental investigations of H. C. Beyer, P. J. Martin, Germain Sée, and others, which seem to prove that in efficient doses it produces a true analgesic effect, manifested by diminished reflex power in the spinal cord, less muscular contractility, and relaxation of the cutaneous vessels, accompanied uniformly, as shown more directly by Martin, by diminished heat production and increased heat dissipation. The paper closes with the following paragraph: "The following conclusions are, I think, justified by our present knowledge of antipyrin in the treatment of rheumatism: (1) it is as efficacious as the salicylate of soda, producing similar therapeutic results, and is less nauseous than the latter, and does not produce headache or ringing of the ears; (2) usually it acts most efficiently in the most frankly acute cases; (3) besides reducing, by its antipyretic properties, the fever and also the pain, which many antipyretics relieve, it reduces the pain by acting directly upon the nervous system." These conclusions appear to have been sustained by the clinical observations of others; but all admit that the treatment with antipyrin does not prevent relapses and cardiac complications in about the same ratio as under the treatment with salicylates.

Salol, another antipyretic recently introduced by Nencki and Sahli, has elicited a greater number of articles in the medical periodicals concerning its value in the treatment of rheumatism, during the past year, than any other remedy. Early in the year 1887, the Berlin correspondent of the *British Medical Journal* communicated to that journal the results obtained by Dr. Herrlich from the use of salol in the treatment of acute articular rheumatism and several other diseases. Dr. Herrlich found it efficient in reducing the temperature and satisfactorily relieving all the symptoms in several cases of the acute form of the disease. It did not, however, prevent relapses in some, and endocarditis in others. It did not produce sweating, like antipyrin, but more frequently caused gastric disturbance; and in one case of chronic rheumatism, in which twenty-four grams (ʒvj) of salol were given in three days, decided symptoms of carbolic acid poisoning were developed. The patient vomited severely for eight days, and her urine contained a considerable amount of phenol. In acute rheumatic fever, Dr. Herrlich gave about one gram every three or four hours, or from six to eight grams in twenty-four hours. The results obtained from the use of salol in the treatment of rheumatism by Von Bielschowsky,[9] of Breslau, are as follow: of twenty-seven acute cases treated by him, nineteen were reported as promptly cured; two resisted the influence of the salol, and yielded to the salicylic acid; while the remaining six continued in a chronic form. Relapses occurred eight times. He gave the medicine in doses varying from two to eight grams (ʒss to ʒij). Four cases developed mild cardiac symptoms that proved temporary. The urine showed the presence of carbolic acid while under the treatment with salol. Rosenberg, of Berlin, reported somewhat less favorable results from the use of salol in rheumatism, and observed all the unpleasant effects that had accompanied the use of the salicylates.

A series of observations and experiments by Dr. Lombard,[10] of Paris, led him to think that the value of salol in the treatment of rheumatism had been over-rated. While its efficiency in controlling the pains was remarkable, the effect was more transient than when salicylate of sodium was used. These experiments also sustained the opinion expressed by others, that the salol was acted upon by the pancreatic and hepatic secretions, resolving it into salicylic and carbolic acids; and unless thus decomposed by the pancreatic fluid it passes through the intestines insoluble and inert.

The facts thus far established, both by experiments and clinical observations, justify the conclusion that salicin, salicylic acid antipyrin, antifebrin, kairin, salol and betol, when given in active doses, exert a direct sedative or analgesic effect upon the sensory, vaso-motor and trophic nervous structures, thereby promptly relieving pain and reducing temperature, while they cause but little change in the chemical constituents of the blood and secretions. Hence their chief efficiency is displayed in the most recent and active stages of rheumatism and in some forms of pure neuralgia. To render the relief in rheumatism more permanent, they should be given in conjunction with soda or lithia to lessen the excess of lactic and uric acids in the system, and such adjuvants for promoting the important secretory and eliminating functions as are indicated in each case.

M. Vulpian,[11] in a communication to the Académie de Médecine of Paris, claims very favorable results from the use of the salicylate of lithia in articular rheumatism, giving it in doses of one gram (grs. xv) four or five times in the twenty-four hours. He regards it as more efficient in relieving the articular pains than the salicylate of soda, more particularly in the sub-acute and chronic cases.

Dr. Arthur J. Dalton, Dr. Walter Greene, and others[12] have reported cases of acute cerebral rheumatism with hyperpyrexia in the treatment of which prompt benefit was obtained by douches and packing and wrapping with ice-cold water. Dr. E. S. F. Arnold[13] recalls the attention of the profession to the efficacy of a specially prepared tincture of colchicum seeds, in the treatment of acute and sub-acute rheumatism. The tincture is prepared by macerating one ounce of the seeds in half a pint of the highest proof alcohol, fourteen days. Of this he gives from fifteen to twenty minims in half an ounce of water every four hours until nausea and active purging are produced, when the acute symptoms speedily disappear. We have seen the same results follow the use of a strong tincture of the seeds many times. It has but little effect in chronic cases. Professor Benedickt,[14] in a paper on "The Use of Subcutaneous Injections of Carbolic Acid," claims to have obtained excellent results from the subcutaneous injection of a two per cent. solution of carbolic acid, in the vicinity of the affected articulations, not only in recent acute cases, but also in many chronic and relapsing ones, and in some where the sheaths of tendons were affected following injuries. The injections were repeated from one to three and four times a day, according to the acuteness of the disease, and were followed by speedy relief from pain and swelling.

The effect did not seem to be limited to the locality where the injections were made, but extended to the system generally as when administered in any other way. Dr. J. Schreiber,[15] in a lecture accompanied by numerous cuts illustrating the required apparatus, claims a great degree of success in the treatment of lumbago in all its stages, as well as chronic rheumatic affections of muscular structures in other localities, by massage assisted by various mechanical appliances. His views and methods are worthy of attention, but his lecture does not contain the results of a sufficient number of cases subjected to his treatment, to enable us to compare them with cases treated by other methods.

GOUT.

Etiology.—Since the important investigations of Dr. Garrod demonstrated the presence of uric acid and urate of soda as etiological factors in the development of gout in its various forms, further investigations have added no new essential or direct causes, but have consisted mainly in efforts to trace the origin of the uric acid and its relations to the processes of assimilation, metamorphosis and excretion, on the one hand; and its *modus operandi* in producing the active symptoms of the disease, on the other. In attempting to trace the successive changes that the albuminoid elements of food undergo in being transformed into blood, it is found that they enter venous blood in the form of chyle; and it is claimed that uric acid is developed at an advanced stage of the process, which by the action of additional oxygen in the lungs, becomes converted into urea, and readily eliminated.

If the production of uric acid should be excessive, or the supply of oxygen deficient, the uric acid would remain in the blood unchanged, and accumulate until it established a specific or gouty irritation in some of the structures of the body.[16]

Whether the undue accumulation of uric acid in the blood in cases of gout results from a deficient supply of oxygen at some stage of the processes concerned in the conversion of food into the natural constituents of blood, as stated above, or from similar defect in the retrograde tissue changes, has not yet been demonstrated.

Dr. J. Mortimer Granville,[17] in a lengthy and valuable paper, accepts the doctrine and the presence of uric acid in excess in the blood as an essential etiological factor directly causative of the local inflammation and deposits. He reasons with some show of plausibility, in favor of his suggestion that the excess of uric acid results from a primary failure of the liver to furnish its normal amount of bile to co-operate with the pancreatic fluid, on account of which the food elements are only emulsified instead of being saponified, as in healthy duodenal digestion. The products of such imperfect digestion being carried through the portal vessels to the liver, deranges its "glycogenic and cognate processes" in such direction as to lead to the "excessive formation of uric acid or the arrest of subsequent processes by which it should be converted or destroyed."

Still another theory of the production of the uric acid in gout is found in a most interesting monograph by Dr. W. Ebstein,[18] of Göttingen. He attributes the production of the greatest part of the uric acid to the muscular structures, aided to some extent by the marrow of the bones. In support of this opinion he refers to the guanine gout in pigs, and to the deposits of uric acid in birds and reptiles, as well as the cases by Neukomm, who found uric acid in the muscles of a girl dead from typhus, and in the muscular structure of the heart of a syphilitic woman. While the results of the investigations of Dr. Ebstein constitute a valuable addition to the previous accumulation of facts regarding the etiology of gout, they do not afford a satisfactory demonstration of the correctness of his views regarding the origin or retention of the uric acid, so generally present in that disease. Both Ebstein and J. Mortimer Granville claim that hereditary influence constitutes the primary predisposing cause of far the larger number of cases of gout in all its phases. The latter declares with emphasis, that the disease "is on the increase and is now affecting classes of the population which were in former times free from its ravages." He also declares that its type has materially changed, presenting new forms and complications, and attacking women much more frequently than in former years. He regards heredity or the propagation of the predisposition to both sexes, as the chief factor in developing the changes just mentioned. It is proper to state that Dr. Granville does not attempt to sustain these opinions by any statistical data or other methods of proof. Consequently they must be regarded as simply the opinions of one possessed of erudition and extended observation.

Pathology and Pathological Anatomy.—As uric acid and urate of soda continue to be regarded as the efficient disturbing agents in the production of the morbid processes constituting gout, so all writers still agree in regarding both the general and local phenomena as the effects of the irritant qualities of these agents retained in contact with the tissues. Many regard the chief symptoms as resulting from direct action of the irritant upon the nervous structures, while Dr. Granville, in the paper to which allusion has already been made, suggests what he calls a new theory. He says "although the nervous system is the prime mover in the production of the phenomena of gout, as it doubtless is in the development of the great majority of functional and nutritive disorders, the exciting, if not also the predisposing, cause of the trouble is to be looked for at the periphery rather than in the centre; that is to say, in this instance, in the liver or kidneys, or both." And he suggests that the primary irritating impression in those organs is transmitted through the pneumogastrics as the afferents to the nerve centres, while the sympathetic serves as the efferent returning the exciting or inhibitory reflex influences, as the case may be, to the organs affected. The chief real addition to our knowledge of the action of the urate of soda and the appreciable changes it induces in the various structures in gout have been made by Dr. W. Ebstein as presented in his monograph. By numerous experiments and dissections he has traced evidences of the direct irritative action of the uric acid or urate of soda, not only in the kidneys or other internal organs, but also in the articular cartilages and fibrous structures, and in the connective tissue between the muscles and below the skin, thereby demonstrating the diffused primary action of the irritant conveyed in the blood. He found in all the structures named some foci of cell or tissue necroses and deposits of urate of soda, and has figured them in the plates contained in his work. As proof that these diffused tissue changes are not secondary or reflex from primary foci in the kidney or liver, he mentions fatal cases of articular gout in which examination revealed no appearance of disease in those organs. He regards the first stage of the disease as "an inflammatory process, set up by the uric acid, leading to foci of necrosis, and then deposits of urates in the cavities thus formed."

Until recently it has been supposed that the lymphatic vessels and glands were rarely if ever involved in gout. At a meeting of the Royal Medical and Chirurgical Society, March 8, 1887, Dr. Debout d'Estices, of Contrexeville, presented (by Dr. Garrod) a paper describing a case of well-marked gout in which the parotid gland became swollen and resisted treatment until gouty inflammation attacked the knee, after which the affection of the parotid quickly disappeared. Mr. S. Paget, Dr. B. O'Connor, and Dr. Garrod reported one case each as having come to their knowledge more recently.[20]

Treatment.—Under this head there is very little to record not previously well known to the profession. Dr. J. Mortimer Granville strongly recommends the drinking of large quantities of pure water for the purpose of holding in solution the urates, and "flushing" them out of the system through the natural channels. He also recommends the persistent internal use of iodine, under the belief that it is capable of chemically uniting with excess of soda, detaching it from its combination with uric acid, and thereby rendering the resulting iodide of soda and uric acid more soluble and easy of elimination. Contrary to the popular ideas concerning the efficacy of certain mineral waters, Dr. Granville insists that the more free the water is from all mineral or saline ingredients the greater will be its efficacy as a solvent of the uric acid and urates, which is undoubtedly correct.

For early relief of acute attacks, Dr. Ebstein recommends the salicylate of lithia as the best remedy. He does not, however, think there is any specific remedy for the cure of gout.

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DIABETES AND DISEASES OF THE SUPRARENAL CAPSULES.

By JAMES TYSON, M.D.,

PHILADELPHIA

DIABETES MELLITUS.

Etiology.—An instance in which diabetes mellitus seems to have been clearly due to the direct action of cold is reported by E. Peiper,[1] of Greifswald. A well nourished girl of 17 years, with healthy living parents, no hereditary tendency to any disease, having had no disease except measles, and who had menstruated regularly since her fifteenth year, became much overheated while dancing, and drank rapidly a glass of ice-cold water. Immediately afterward she was seized with excessive thirst, and drank in vain large quantities of water. On her return home, she became intensely hungry, and passed freely urine of a clear, straw yellow color. Thirst and copious urination continued day and night, and during the ensuing month she began to lose strength, was easily fatigued, and finally could do no work. Her weight diminished, and when admitted to the hospital she was of spare habit, skin cool, muscular system moderately developed, panniculus adiposus wasted. The tongue was coated, the visible mucous membrane reddened. Her appetite was now comparatively impaired, her stools delayed. There was no derangement in the special senses, nor pain in the head or neck. The urine, of which there were 4000 to 5000 ccm. in 24 hours, was acid, straw yellow, and contained a considerable amount of sugar, but no albumin. The other organs appeared normal. After 8 weeks' treatment the sugar had disappeared, and her weight increased 7 pounds. Peiper relates another case, that of a farmer, who stood for an hour in water as far as his knees, and was suddenly seized with a thirst so intense that he snatched a crock of water and drank the whole at once. Profuse urination followed promptly, and in the course of a few days, other symptoms of diabetes.

C. J. Kelly[2] reports a similar case. A boy, 10 years old, previously perfectly healthy, drove a cow, on Nov. 26, a distance of two and a half miles. By his exertions in trying to keep the animal on the right road he got into a great state of perspiration. Feeling thirsty, he stopped at a roadside pump and took a great drink, thinking he could never get enough to quench his thirst. This symptom with copious micturition, continued until he died Dec. 11, eleven days after the symptoms set in. Peiper further says that out of 46 cases treated at the Greifswald Polyclinic during the year, eight could be ascribed to this cause, most of the patients following sea-faring occupations. The influence of cold as a cause of diabetes is denied by many. Cantani will not acknowledge it to be either a predisposing or an exciting cause, although he admits that in 6 out of 218 cases observed by him there is a possible relation, other possible causes existing, however, in every case. Griesinger, on the other hand, would ascribe 40 out of 152 cases to cold or a thorough wetting of the body. Cases are also reported by Kulz, Zimmer, Opholzer, Frierichs, and Roger. In none of these cases, however, was there the direct sequence exhibited by Peiper's and Kelly's cases.

As to cases of supposed nervous origin, David Drummond[3] reports one, a boy aged 7, who was admitted into the infirmary, greatly emaciated, complaining of undue thirst and other symptoms of diabetes. It was stated that he had received a blow on the head some five months previously, and that he had suffered ever since from headache. The urine was full of sugar. He died in a state of diabetic coma two days after admission. [For autopsy of this case see *Morbid Anatomy.*]

M. S. Falls,[4] of Washington, considers the emotional factor of much more consequence in the etiology of diabetes than is commonly supposed, and that it is the most frequent of the long list of predisposing causes named by writers. He believes it to be due to the large number of centres concentrated in the medulla oblongata, in addition to the diabetic centre and in proximity to it. The almost continuous use of one or more of these is accompanied by a hyperæmia, which is propagated through continuity and contiguity to the diabetic centre. If now we add to such hyperæmia the effect of powerful emotions, such as prolonged shame, fear, chagrin, anxiety from any cause, the effect is increased and diabetes results. Two cases are related by him in illustration, of the nature of those usually quoted in favor of this view. O. Kohler[5] collected 21 cases of polyuria after brain injuries, and 21 cases accompanying non-traumatic encephalic disease. From these and one of each kind occurring in his own practice, he concluded that polyuria always indicates some affection of the parts of the brain in or near the posterior fossæ, the cerebellum, pons, or medulla oblongata, but that more exact localization is not justifiable. Experiments on animals showed that similar changes in urine accompanied lesions of the medulla oblongata, the pons and cerebellum, but that the vermiform appendix has no special connection with any morbid change in the function of the kidneys. Polyuria was caused most frequently when the lateral parts of the posterior end of the pons and medulla were injured. The polyuria indicates some positive lesion, transient or permanent, of the parts concerned, and is never a mere functional disturbance. If it be a transient hyperæmia that causes it, the symptom will be transient; if, however, the symptom is permanent, we must conclude that there is some organic lesion of the same part.

Seegen,[6] of Vienna, says that of the two forms of the disease, mild and severe, as classified by him, 90 per cent. of both are due to some disturbance of the nervous system. He excludes, however, those severe nervous affections sometimes accompanied by diabetes. In many families where diabetes is hereditary, certain members, psychically ill, will suffer from melancholia or a disposition to self-destruction, while others will be diabetic. Other cases are due to depressing emotions, great anxiety or shock from sudden misfortune.

Additional attention is called by Arnaud,[7] in a recent thesis, to the fact that syphilis may cause diabetes, the latter developing after syphilis and seeming to be the direct or indirect result of the disease. This may be owing to changes in the blood and tissues, due to syphilis or to the development of gummata or other tertiary products in the parts surrounding the fourth ventricle, thus encroaching upon the glycosuric centre.

Under the head of etiology may be included observations upon the so-called lipogenous diabetes, by E. Heinrich Kisch,[8] and Hardy.[9] In general lipomatosis, examinations of the urine not infrequently discover temporary glycosuria, separated by considerable periods of its total absence. In addition to this, however, Kisch discovers diabetes to be a very general consequence of lipomatosis, especially when the latter is hereditary, develops early in life, or progresses rapidly. In more than half of the cases of hereditary lipomatosis of high degree, Kisch has found diabetes mellitus to develop; while in the other forms, about 15 per cent. acquire diabetes. It sometimes happens that certain members of some families are very fat, even in early life, while others, without becoming especially fat, acquire diabetes; or there may be several members of a family who in early life exhibit a disposition to large accumulation of fat, and between the ages of 30 and 40 become diabetic.

As to the relation between general lipomatosis and diabetes, Kisch says it is reasonable to believe that the same defects of diet which lead to one cause the other. Among these may be mentioned starchy and sweet foods. Lack of exercise may also coöperate, though this is true only of a certain number of cases. If the accumulation of glycogen in the liver is the prelude to fat formation, it is easy to understand why, in fat persons the disposition to diabetes is great. In fat persons, there is also a certain pressure upon the liver, as the result of which, according to Pavy, glycogen passes rapidly into the blood, and is converted into glucose. There are united, therefore, in the fat person, two predisposing causes,—abundance of amyloid substance and pressure of the abdominal wall on the liver.

Zimmer has also sought in conditions of the liver an explanation for this tendency to diabetes. In fat persons the secretion of bile is more or less unfavorably influenced, while the separation of glycogen continues. Further, when glycogen and fat are accumulated in the liver cells, their conversion and oxidation are limited, until finally the sugar which the blood of the liver carries to it, passes directly through it without being converted into glycogen.

Kisch, however, does not believe that the fatty liver can be held responsible for the relation between general lipomatosis and diabetes; for if the fatty liver could be the cause of diabetes, the two would be found more frequently associated. He would rather call attention to the alteration of the muscles, their interpenetration with fat and the consequent separation of the muscular fibrillæ, fatty degeneration of the latter, as well as the influence exerted upon muscular exercise thereby, as an explanation of the connection between lipomatosis and diabetes; and concludes that lipogenous diabetes and general lipomatosis are the result of a congenital abnormality of tissue cells, by reason of which in the one case the fat is insufficiently burned up, and in the other, sugar is not consumed as in health.

In this country there does not seem to be that close relation between lipomatosis and diabetes claimed by Kisch. Fat persons early in life are no more disposed to diabetes than others. Past middle life, it is well known that glycosuria is found in many stout persons, and a certain number of them "lose flesh," but for the same reason that other diabetics emaciate. The carbohydrates, instead of going to form fat and contribute to force production, are excreted as glucose. These stout persons are prone to acquire glycosuria because they are apt to be free livers, and to lead indolent lives; and their glycosuria is apt to be of the alimentary kind. For this reason it is very easily controlled by diet, while the patient may live on for years with health slightly, if at all, impaired.

Nature and Pathology.—The most original conception as to the nature of diabetes mellitus is contained in W. Ebstein's[10] new book on this subject. According to him, diabetes mellitus is not a symptom of different diseased states, but an independent disease due to a defective state of the protoplasm, in consequence of which too little carbonic acid is produced in the tissues. As a result of this anomaly, the diastatic ferments contained in the organism act too strongly upon the glycogen contained in the different organs, converting it into the easily diffusible varieties of sugar, which are absorbed and partially excreted. In severe cases, also, the albuminous matters are similarly attacked by the ferments and converted from a solid to a liquid state. The author claims to have shown by extensive experiments that carbonic acid has the power to inhibit the action of the diastatic ferments, especially upon glycogen. If now from any cause the protoplasm loses the power to regulate carbonic acid production, the glycogen contained in the organism is defenceless against the action of the saccharifying ferment, and there arises glycosuria. While in the normal organism sugar formations and sugar consumption in the tissues balance each other, in that of the diabetes this balance is lost, so that the easily diffusible sugars are produced and the unused portions are left over for excretion.

While now in certain cases this deficit is made up by an increased amount of albuminous food (which also produces carbonic acid), there are others in which the derangement of the oxidation processes in the protoplasm is so considerable that the carbonic acid arising from the nitrogenous foods is not sufficient to protect the glycogen, so that the albumin of the body must be drawn upon to make up the carbonic acid deficit. These furnish the severe forms of diabetes. As a consequence of diminished carbonic acid formation, less oxygen is used, whence follows lowered body temperature,—an extremely unfavorable symptom.

The albuminuria which after the glycosuria, in severe cases, is the most important symptom, Ebstein also ascribes to deficient carbonic acid formation on the part of the protoplasm; for the carbonic acid also protects the slowly diffusible globulin and prevents its conversion by the ferments of the organism into the easily diffusible albuminous matters.

The extreme thirst Ebstein ascribes in part, as others do, to the abstraction of water from the tissues by the sugar circulating in the blood. Again, glycogen is not converted, as in the healthy organism, into carbonic acid and water, but remains in the shape of carbohydrate, so that in this way the tissues are again deprived of a portion of water.

As to pathogenesis, Ebstein believes we have probably to do with a congenital defect of the protoplasm above alluded to, whose symptoms are excited by some accidental causes. These arise, among the poorer working classes, from a too early use of the organs of the body; among the rich in opposite modes of living, rest, etc. Ebstein is very skeptical with regard to the etiological relation of brain affections, whose causal relations to diabetes he regards as far less than is commonly supposed. The transitory glycosuria produced by puncture of the fourth ventricle is something altogether different from chronic diabetes, for the explanation of which it is absolutely insufficient. The brain alterations may with greater propriety be regarded as consequences rather than as causes of the diabetes, and the same is true of the alterations of the semilunar ganglion and of the pancreas, to which, according to Ebstein, altogether too much significance has been assigned in the causation of diabetes.

These theoretical views as to the nature of diabetes lead the author to the conclusion that fat is not only admissible, but strongly to be recommended as a diet, since fat is oxidized in the organism directly into CO_2 and water, and furnishes to the diabetic those combinations through whose want the characteristic derangements of tissue change arise. Fat is of course to be given in different shapes. Nor does Ebstein favor a pure flesh diet, but permits his patients 60 to 100 grams of bread a day, the substitutes for which he rejects on account of their greater or less indigestibility. He further seeks to increase carbonic acid formation in the tissues by muscular exercise, active and passive. The action of the Carlsbad waters he ascribes to their abundance of carbonic acid.

Scarcely less important is the latest contribution of J. Seegen,[6] already referred to, who has supplemented his large experience with diabetes by extensive experiments, repeating, among others, those of Bernard and Pavy. He reaches the following conclusions: (1) The sugar formation is a normal function of the liver, and goes on without interruption. (2) The amount of sugar formed in the liver every day is very large, hundreds of grams passing over into the circulation daily from this source. (3) The sugar found in the liver is continually being decomposed in the body, although in what precise parts is not perfectly clear. (4) And here we have the first radical difference between Seegen's conclusions and Bernard's: the sugar and other carbohydrates taken in with the food take no part in the sugar formation in the liver. (5) On the other hand, albumin and fat are the only materials out of which the liver makes its sugar; while (6) the sugar and starch taken in with the food are converted by the liver into glycogen, which is, however, not reconverted into sugar. Thus the liver is both a sugar former and a glycogen former, the sugar being produced out of albumin and fats, and the glycogen mainly out of the food carbohydrates. Of these six conclusions he is absolutely certain.

Clinically he makes two varieties of diabetes, a first in which the subjects are generally well nourished, and often of good color, and moist skin, while there is no excessive appetite, thirst or urinary secretion. In the second the patients rapidly emaciate, become pale, muscular power rapidly disappears and hunger, thirst and urine are excessive. Subjects of the first form are between 40 and 50 or older, in the second and children or adults up to 25. In the first form the patients pass diabetic urine only when they take the carbohydrates with their food, the liver-cells being unable to convert them into glycogen. In the second glycosuria continues regardless of the food. These two he regards as distinct forms of disease rather than as stages of the same disease although he will not deny that the first may pass over into the second.

Dr. Pavy,[11] before the Ninth International Congress, reasserted that the immediate cause of glycosuria is an insufficiently dearterialized venous blood, which in turn is due to a vaso-motor paralysis and dilatation, especially of the vessels of the chylopoietic viscera.

A contribution to the action of phloridsin in producing diabetes, and which may be of signal value, has been made by von Mering,[12] of Strasburg, to the 6th Congress of German Physicians. If a dog is kept without food for two days and then given phloridsin, its tissues become free of glycogen in 48 hours, none being demonstrable in liver or muscles. A dog weighing 20 kilograms, after 3 days' fasting and the ingestion of 20 grains of phloridsin, excreted 75 grams of sugar; after seven days' fasting and ingestion of 20 grams at six o'clock in the evening there were secreted the next morning at 10 A.M., 125 cc. of urine with 10 per cent. of sugar; at 3 P.M., 80 cc. of urine with 15.9 per cent. of sugar and 5.4 of urea; at 6 P.M., sp. gr. 1097, 19 per cent. of sugar and 8.5 per cent. of urea.

The amount of sugar secreted in one day was 31 grams. On the twentieth day were secreted, without ingesting carbo-hydrates, 19-5 grams, such a quantity as occurs in the severest cases of diabetes. As no carbo-hydrates were ingested, the sugar must have been derived either from the muscles or fat, according to von Mering from the muscular tissue only, because neither in diabetes mellitus nor in phloridsin diabetes does the ingestion of fat increase the sugar. It is well known through the researches of Pettenkofer and Voit, that a diabetic patient consumes less oxygen and excretes less carbonic acid, the sugar refusing to be oxidized. Von Mering shows, too, that sugar is not produced from fat; therefore it must come from albuminous tissues. The latter is shown by experiment, and is confirmed by a glance at the extent of the carbo-hydrate combinations in albumin. He also shows that the sugar cannot come from the phloridsin. Albumin is to be considered as a glucoside which produces carbo-hydrates. In the organism, fat is formed out of carbo-hydrates and albumin. It is conceivable, therefore, that out of the disintegration of fat, sugar may be formed. According to von Mering, the nature of diabetes depends upon the incapacity either to store up sugar as glycogen, or to oxidize it. Conspicuous in connection with these experiments is the fact that the quantity of urine was not increased. To test the action of phloridsin, von Mering took some himself, and found that the desire for sugar was diminished; otherwise it did him no harm.

Symptomatology.—The symptomatology of diabetes mellitus has been enlarged during the past year by the closer study of certain derangements of vision. Hirschberg[13] has ascertained that the most frequent of these is a defect in accommodation. Three forms are distinguished: in the first and mildest, if the patient is not older than 45 years, he either needs no glasses at all, or those adapted to his eyes are sufficient to enable him to read the finest print. There is, however, no power of endurance. The condition is like the weakness of accommodation succeeding an exhausting illness. The second form is a true paralysis of accommodation without enlargement of the pupil, as after brain pressure. Patients with such degree require stronger glasses than those corresponding to their age, and with them the endurance is also wanting. The third form is even more serious, and presents itself in the shape of a sudden occurrence of dimness of vision. This form was earlier regarded as amblyopia, but as a defect of accommodation and not of the optical apparatus. Suitable convex glasses cause the dimness to disappear. The affected patients are far sighted and at first can apparently overcome the difficulty in refraction by thickening their glasses; but this cannot be done after the paralysis of accommodation has set in. Several illustrative cases are related by Hirschberg in which the symptoms promptly disappeared after adopting a suitable diabetic treatment.

A second very important form of visual defect is diabetic amblyopia. First described by Griesinger, it was thrust into the background by the discovery of the defects of accommodation, so that five years previous to the preparation of Hirschberg's essay, but five cases were reported; while Hirschberg himself adds seven carefully observed cases. The subjects are unable to see small objects, and use increasingly stronger glasses for reading, but even these are found insufficient. The eyeground is normal, but while the periphery of the field of vision is normal, there is a dark spot in the middle. This either surrounds the fixed point or borders on it. In contrast with the defect of accommodation first described, this diabetic symptom indicates an unfavorable prognosis, not merely as to the prospect of recovery, but as to life; for out of Hirschberg's seven cases, four died in about a year, and the fifth sooner. One died suddenly of diabetic coma the day after admission to hospital. Among visual complications accompanied by alterations in the eyeground, are occasionally suppurative keratitis and iritis, with fibrinous exudate. The latter usually depends upon syphilis or rheumatic arthritis, and when these are not present, the urine should be examined. The most important structural change, and one which is comparatively common, affects the crystalline lens.

While Arlt, as far back as 1863, discouraged all cutting operations where the lens is changed, von Græfe operated frequently on diabetics without unfavorable results; and Hirschberg himself, in 20 extractions, had a single unfavorable result. The law laid down by Jüngken, that eye operations should never be performed in dyscrasic affections until the latter are removed, is not applicable to diabetic cases. Cataract operations in diabetic patients should be avoided if possible, but if there is no vision and the patient desires to recover sight, the operation should unhesitatingly be done; for if cataract is once fully established in a diabetic, it is not possible to remove it by internal treatment, even though the general symptoms of the diabetes may be relieved. The cataract continues to ripen. The prognosis of the operation is good, although the patient does not always attain as perfect vision as that which immediately succeeds it. When cataract occurs in diabetics of advanced years, its course is generally similar to that of the same age in which cataract is independent of diabetes.

Very delicate striæ can often be seen in the crystalline lenses of diabetics, but these are quite compatible with good vision, and can be directly controlled by treatment. Very characteristic is a form occurring in young diabetics with sunken faces and sharp noses, which may develop in a single week. As to the vitreous body, it is subject to cloudiness which correspondingly obscures vision, but which, by the usual means of treatment, disappears and vision is again restored. The retina is relatively frequently involved,—out of 24 diabetics, 6 times. Two principal varieties are observed. The first consists of small circumscribed bright foci, arranged in groups in the middle of the retina. The second series is hæmorrhagic, and include two subdivisions. There may be hæmorrhagic infarct of the entire retina, with total loss of vision, and the entire vicinity of the entrance of the optic nerve is filled with blood. The prognosis is quite unfavorable. Much less serious are isolated hæmorrhages, found in the periphery as well as in the vicinity of the optic nerve. Sometimes dark spotted foci are observed, especially in old diabetics, if observation is continued long enough. Retinitis similar to specific retinitis, or retinitis pigmentosa, is very seldom seen. One case Hirschberg diagnosticated only with the aid of the ophthalmoscope, which revealed small, bright, shining foci in the retina of a man of 50, with 3.6 per cent. of sugar, followed by gangrene and death. These observations are important because they support the view that hæmorrhages may occur elsewhere in the nervous system, where they are not visible. As to the affections of the optic nerve, they are very varied. Most important is an axial neuritis, which effects an atrophy of the bundle of nerves occupying the centre of the optic nerve. The symptoms are similar to those of diabetic amblyopia. In the middle of an otherwise clear field of view, there is a dark spot. There occur also various defects in the periphery of the field of vision, as for example, half vision. Some of these disappear with good recovery. Paralysis of the muscles of the eyeball is also to be included in the defects of vision belonging to diabetes. Among these is suddenly occurring binocular diplopia, and in any case of sudden diplopia where the cause is not plain, the urine should be examined. In some cases sugar may be found, in others albumin. The usual form is paralysis of the abducens, although the oculo-motor may be paralyzed; and sometimes there are combined forms, or an alternation of the paralysis. Even the appendages of the eye must not be overlooked. Thus, in one case there were small furuncles of the lids extending over a period of nine months, and 1½ per cent. of sugar was found. Impaired hearing due to swelling and œdema of the Fallopian tube is included by Miot[14] among the symptoms of diabetes.

Under the head of genital diabetes, Fournier[15] describes two forms of eczema affecting the genitals in diabetics. In women there is first an erythema which commences generally in the neighborhood of the meatus urinarius, and diffuses itself thence upon the vulva. Then in most cases succeeds an eczema, acute or chronic. The acute form is characterized by a redness and swelling, and an abundant discharge, which soils the linen, a painfulness in walking, and above all, an intense and continuous itching, with exacerbations. Sometimes it precedes all other symptoms of diabetes, as evidenced in a patient of Fournier's, a lady thoroughly familiar with the symptoms of the disease from having nursed a diabetic uncle through a long illness, and who was perfectly astonished when informed that her urine was saccharine. This affection, relieved by treatment resulting in a removal of the sugar, is apt to return with the reappearance of the sugar, so that one may predict the latter from the recurrence of the former.

The symptoms of the second or chronic form are those of chronic eczema. There is noted a somewhat wine-colored redness of the discharge or desquamation, and the itching is abominable. This causes extreme nervousness and sleeplessness, and finally loss of strength and appetite, indigestion and emaciation. At length there results such a thickening and hardening as to justify the name pachydermatous eczema. Finally cryptogamic patches a few millimetres in diameter make their appearance in the larger and smaller labia and the folds between them. In men there exist, in connection with the glans penis, three forms, erythematous balanitis, a herpetic balanitis and an eczema. The first consists in a partial redness of the glans immediately surrounding the meatus; the second is characterized by a series of reddish islets; and in the third, the surface of the gland is granular and fissured. Whenever, therefore, these symptoms present themselves, the urine ought to be examined. The prepuce also may become inflamed, thickened and parchment-like, and fissured when the patient attempts to retract it.

There also occurred in Fournier's clinic[16] a case of spontaneous gangrene of the penis in diabetes, which was at first regarded as possibly a syphilitic gangrenous chancre, or gangrenous gumma, but the discovery of sugar cleared up the case.

Nervous Symptoms.—The appearance of glycosuria in such diseases as multiple sclerosis, locomotor ataxia and other affections of the nervous system, indicates the involvement of that part of the floor of the fourth ventricle presiding over sugar formation, and is generally an unfavorable symptom, especially if sugar is abundant. This is well shown by a case related by Edwards.[17]

The disappearance of the knee-jerk in diabetes has been a symptom sought for and recognized since the observations of Bouchard. More recently Marie and Guinon[18] and Raven[19] have published confirmatory observations. The former have particularly called attention to a source of error in diagnosis in the simultaneous presence of other symptoms of tabes, such as abolition of reflex movement, lightning pains, slight staggering when the eyes are closed, gradual loss of sight and sexual power and difficult micturition. Notwithstanding these symptoms, tabes may be excluded if glycosuria is present. The absence of the knee-jerk does not indicate any special form of diabetes, but simply that the disease has entered a more serious phase, as shown by the following: out of the first series of 66 patients, 47 retained the power of reflex movement, while 19 had lost it. Of these 47 cases 2, or 4.25 were fatal. Of the 19 who had lost the power, 6, or 30.3 were fatal. In another series of 111 patients, 70 possessed the power of reflex movement and 41 had lost it; of the 70 cases, 5, or 7.14 proved fatal; while of the 41 cases in which the power had disappeared, 7, or 14.63 were fatal. That is to say, the mortality in the cases in which the power of reflex movement had disappeared, was more than double that in the others. Raven reports a case of improvement in the other symptoms, accompanied by reappearance of the knee-jerk. Dreyfus[20] goes so far as to draw a line between cases of glycosuria which are attended with a diminution of the knee-jerk, and those in which it is exaggerated, and excludes the latter from the category of true diabetes. True diabetes he regards as a depressing disease of the nervous system, and especially of the spinal cord; and when along with glycosuria we find the tendon reflex exaggerated, it may be declared that the case is not diabetes, but a simple "glycosuria." Jendràssik[21] says the knee-jerk can be revived in cases of diabetes and neurasthenia, where it has sunk to almost nothing, by making the patient take a muscular effort with the body and arms, while the knee-jerk is under examination.

Pavy[22] called attention to the red tongue in very severe forms of diabetes. This he considers due to the dilated condition of the vessels of the mouth, which is extended to it from the vessels of the chylopoietic viscera, which are also dilated in such cases, and in paralysis of the vaso-motor nerves.

Relation between Gout and Diabetes.—J. Mortimer Granville[23] has recently pointed out the characteristic symptoms of "gouty diabetes," or, he says, "more accurately *undeveloped gout*, in which the symptoms are chiefly and often characteristically diabetic." Persons affected are generally of large frame, with little stability or power, full sized but flabby muscles, and a tendency to the accumulation of fat. They are great, or intermittently great eaters, energetic or impulsive, but weak and easily exhausted. Their skin is pale, their extremities cold, they sleep badly at night, and worry in their wakefulness; while by day they are drowsy and falling asleep, breathe heavily, or if not roused, sink into a state verging on coma, with puffing, sighing or labored respiration. They are subject to attacks of vertigo, in which objects seem to roll from left to right. They have often weak hearts with irregular pulse, and perhaps a short mitral systolic murmur, not constant, and more likely to be heard when the heart is acting languidly than when recently stimulated by exercise. The characteristic phenomena are (1) great variability in the quantity, color, and density of the urine, and the want of relation between the bulk of fluid ingested and that excreted. (2) Sugar is present when the urine is small in quantity rather than when abundant. (3) The acidity is abnormally low in proportion to the percentage of urea. (4) There is generally much uric acid when the sugar is absent and less than the average in health when sugar is present. (5) Albumin is often found, soon after a meal, either with the sugar or during a free elimination of uric acid, and when there is no albumin the phosphates are generally in excess. (6) The liver is enlarged vertically, the bowels constipated, with occasional attacks of diarrhœa, stools pale when scanty, and bilious when profuse. Hæmorrhoids are usually present, and when they do not bleed they often attain great size. Not infrequently there is a prostatic gleet.

Diabetic Coma.—The importance of a more correct understanding of diabetic coma will be appreciated, when it is recalled that according to Frerichs, out of 250 cases of deaths 153 are due to coma. It will be remembered that diabetic coma is ascribed by von Jaksch to the presence of diacetic acid in the blood. Observations by Stadelman, Kultz, Menkowski, Lépine and Hugounenq, go to show that the substance in the blood which is responsible for the symptoms of diabetic coma, is β oxybutyric acid. Hugounenq[24] had an opportunity to study a case dying in diabetic coma in the clinic of Lépine. He examined first the urine of the patient, before any treatment was instituted; second, the blood drawn before a solution of 40 grams of sodium carbonate was injected into the veins; third, the urine after this injection. In the first specimen he found β oxybutyric acid in addition to sugar. In the second, the blood, he found the same acid. The third specimen contained neither the acid nor sugar. Thus was established the presence of β butyric acid in the blood of the patient having diabetic coma; and its disappearance coincidentally with the intravenous injection of large quantities of sodium carbonate. In explanation, β oxybutyric acid is the homologous superior of lactic acid of the muscles, that is to say it differs from the latter only by containing one atom more of carbon and two of hydrogen. Its constitution is further analogous to that of lactic acid of muscle in that it presents all the rest of its properties. It is a syrupy substance, uncrystallizable, affecting polarized light like lactic acid; but while the latter has a dextro-gyratory power of 3.5 degrees, β oxybutyric acid turns the plane of polarization to the left 23.4 degrees. From this, the formation of β oxybutyric acid, at the expense of diseased tissue, is no more remarkable than the production of the latter acid in wearied muscle. Diacetic acid is but a further oxydation, and is in fact found in the urine with β oxybutyric acid. Diacetic acid is, however, very unstable, and readily changes into acetone, the last of the series. By just what process in the living body sugar is converted into β oxybutyric acid and thus the change from glucose to acetone completed, is not understood. Outside the body, there is a simple method by which the change may be brought about, the glucose becoming alcohol, aldehyde, and then aldol, before it reaches the condition of oxybutyric acid.' The same result is completely attained, ultimately, in the body, whatever the method may be. These studies show us how, in the diabetic dyscrasia, glucose or the various compounds derived from it, leave the body before being completely consumed into water and carbonic dioxide, as in health. Thus the organism loses the heat of their combustion, a part of the chemical energy from which it draws its strength. Rational therapeutics teaches us, then, to attempt to consume these substances in the economy. (See *Treatment.*)

In connection with this point it should be stated that, as far back as 1883, Ralfe, before the Pathological Society of London, first drew attention to the fact that the symptoms of diabetic coma were not unlike those in animals poisoned by the injection of acids into their veins, or when attempts were made to diminish the alkalinity of the blood by other means; whence Ralfe inferred that the poison was of an acid nature. The idea of acid intoxication is confirmed by the more recent studies referred to.

In our efforts to determine the relation between acetonuria and diabetes, it is to be remembered that there may be diabetic coma without acetonuria, that there may be acetonuria without coma, and even without diabetes.

Jaccoud[25] distinguishes first a typical form of diabetic coma, with two distinct periods. At first the symptoms are mostly abdominal, including especially digestive troubles, vomiting, with constipation, meteorism, pain in the abdomen, sometimes general and sometimes localized, especially in the right hypochondrium. These phenomena of the first period resemble at the outset those of peritonitis; but an important difference is that the temperature is below normal. With the disappearance of these symptoms the patient becomes apathetic, later drowsy, and finally enters into coma. The second period thus established, lasts for a time ranging from 24 to 48 hours, when the patient dies with a subnormal temperature of 36° to 25.5° C. If we put down three days for the abdominal stage, and two for the coma, five days will represent the mean duration of this period; but it is to be remembered that coma may kill in a much shorter time, in 36 and even 24 hours, and that consequently this duration of five days only applies to the more prolonged cases. The larger number of patients who fall into this stage exhale the odor of acetone.

The second form is altogether different, not in its termination, the coma, but in the absence of abdominal symptoms and dyspnœa. Instead, there are headache and dizziness, which, after lasting some hours or half a day, are followed by somnolence, coma and death.

There is also a third form, the rarest of all. It, too, terminates in coma, but the phenomena which precede it are entirely different. The first symptom is one of extreme fatigue, bordering on total want of power, accompanied by increasing feebleness of the pulse and coldness of the extremities, with cyanotic tinge, more or less pronounced, of the face and extremities, terminating finally in somnolence and coma.

In a review of the symptoms of diabetic intoxication Bernheim and Simon[26] furnish the following summary: (1) The digestive functions are often the first affected. In two of their observations were found vomiting, eructation, pyrosis, epigastric pain and hypochondriasis. (2) Respiratory troubles consisting in dyspnœa without appreciable pulmonary lesion, evidently nervous. This dyspnœa, sometimes anticipating and sometimes simultaneous with cerebral excitement, appears to be a constant phenomena in diabetic coma. (3) Cardiac symptoms very marked in two cases, along with cyanosis, lowered temperature, feebleness of pulse and collapse. (4) Among cerebral symptoms there are delirium, excitement, convulsions and finally coma. Coma is often early, succeeding very rapidly epigastric pain; sometimes, on the other hand, it does not appear till the close of the scene. To these may perhaps be added irritation of the peripheral nerves, explaining the neuralgia so frequent in diabetes. On the other hand spinal symptoms are very unusual.

Complications.—A. Pollatschek,[27] of Carlsbad, has published some statistical results of the systematic examination of diabetic urine for albumin. He found that out of 1187 specimens containing sugar, ranging in amount from traces to 5 per cent., there was more or less albumin in 437 or 37 per cent., which was almost the same as in cases examined in 1885. No constant relation was found between the amount of albumin and sugar, the smallest number of albuminurias, 29.1, occurring in cases with a minimum quantity of sugar; and the highest, 43.8, in urine having from 2 to 3 per cent. of sugar.

As a result of his experience with 8 cases, Reynier[28] has recently called attention to the state of the tendon reflex in diabetes, as a valuable prognostic sign by which to judge of the propriety of surgical operations. Indicating as it does a decadence in the general condition of the patient, its absence may be regarded as contra-indicating operation, while its presence would be regarded as favorable. M. F. Tarnier, while he regards the facts communicated by Reynier as very interesting, thinks that operation in most cases must depend upon the exigency of the case.

Koenig[29] points out that diabetic patients are subject to a low form of inflammation; that the tissues present a favorable nidus for the development of micro-organisms and show a ready disposition to necrose. In all cases of spontaneous gangrene the urine should be examined for sugar, and in surgical complications of diabetes, the first and most persistent treatment should be anti-diabetic. Amputation should not be entertained until diabetic symptoms are retrograde, except as a *dernier ressort*,—a possible means of saving the patient's life.

It has long been known that syphilis may be a cause of diabetes, but the effect of diabetes upon syphilis has claimed less attention. In a recent thesis, Arnaud[30] calls attention to the fact that a patient may have been suffering from diabetes for a longer or shorter time when he acquires syphilis, when the latter complaint is modified in its evolution and progress, from the coincident presence of diabetes. When syphilis develops in a diabetic patient, infective chancre and secondary lesions have a tendency to ulcerate, and the evolution of syphilis appears more rapid than in an ordinary case; and as the lesions deviate from their ordinary course, they may give rise to an error in diagnosis. According to some writers, sugar disappears when specific symptoms show themselves, and Arnaud's observations prove that sugar leaves the urine much more rapidly than in ordinary diabetes, while syphilis is more serious in diabetic cases.

H. Fink[31] regards it as probable that in diabetes we meet with two forms of pulmonary phthisis: one an ordinary tubercular process which does not differ from the ordinary forms of the disease met with in persons not affected with diabetes, and the other the so-called fibrous pneumonia, which clinically is especially distinguished from the former by the absence of tubercle bacilli in the sputum, in its pathological anatomy, in the absence of tubercular or caseous changes, but characterized by a chronic induration of the diseased portion of the lung. Fink gives the clinical history and the results of an autopsy in a case, which go to confirm his view.

As unusual interest and importance always attaches to cases of children with diabetes mellitus, we give the following list of those published during the year: T. McCall Anderson,[32] two cases in children 2½ years old, one fatal within six weeks from the onset of the illness, the other nine days after the onset. C. J. Kelley,[33] a boy of 10, died 16 days after appearance of symptoms. Wm. Frew,[34] of Kilmarnock, girl, aged 9 years. Nine months after she was first seen double cataract developed. This was operated upon, but she died a few days later, about 18 months after she came under observation. Albert F. Fuchs,[35] boy 4 years and 11 months, died in coma the third day after first seen. J. L. Prevost,[36] girl 6½ years old when first seen, died in coma a year later. The editor of this department had under his observation for eight months in the past year a little girl who, when first seen by him was 4⅓ years old, and who died at the expiration of this period more immediately from the effects of diarrhœa induced by eating a large quantity of "macaroons,"—a sweet cake containing almonds. During the entire period until shortly before her death, although emaciated and bloodless, she changed very little under a diet only partially restricted and a drink consisting of sodium arsenite and lithium carbonate in the proportions named elsewhere. So harassed was she night and day by the symptoms and so difficult to manage, that the family finally, in a state of desperation, allowed her to have almost any thing she wanted, and the diarrhœa was the immediate cause of death at 5 years of age. The quantity of urine ranged from 65 to 200 ounces per diem, sugar from 15 to 34 grains per ounce and the specific gravity from 1027 to 1040. The editor also had under his observation for two months a boy of 13.

Morbid Anatomy.—Observations going to show the morbid anatomy of diabetes continue to be few. McCall Anderson[37] alludes to the case of a child 2½ years old, in which an autopsy by Coats and Stevens revealed nothing but slight anæmia of the brain, and some fatty change in the renal epithelium, such as might be accounted for by the intense diuresis.

At the autopsy reported by Drummond of a boy who had glycosuria and who died of chronic meningitis, after injury to the head, the body was found greatly wasted, and post-mortem rigidity very pronounced. The stomach was considerably dilated, and its walls were very thin. The liver was pale, but a large quantity of blood poured out from the cut veins. The dura-mater was strongly adherent to the upper part of the occipital bone. There was a slight amount of arachnoid effusion. The pia-mater beneath the cerebellum was much thickened and congested, especially the portion forming the roof of the fourth ventricle. The lateral ventricles were dilated and contained fluid. The *iter e tertio ad quartum ventriculum* was considerably dilated, and the usual smooth surface of the floor of the fourth ventricle presented a large number of smooth, glistening elevations, the whole resembling the ground-glass appearance of the ependyma so characteristic of general paralysis. A small hæmorrhage occupied the central sulcus, 4 mm. above the lineæ transversæ, and 16 mm. above the calamus scriptorius.

In an autopsy by E. Cless[38] on a diabetic woman who died from the effects of an accidental fall on a smooth floor, there was found, in addition to the brain hæmorrhage which caused her death, an enlarged liver containing three walnut-sized foci of multilocular ecchinococcus. The kidneys were also $\frac{1}{3}$ enlarged and the seat of chronic parenchymatous nephritis.

Prognosis.—Hardy[39] considers definite cure possible, even though rare. He has even seen recovery follow diabetic coma, which he considers, on account of the frequently accompanying albuminuria, of uræmic origin. Neither are grave complications and intercurrent diseases always fatal. Twice he has seen persons with diabetes recover from severe attacks of pneumonia. As a general rule, the complications of diabetes are not marked by any peculiarities in their course. Only gangrene of the lungs is always fatal, and runs a rapid course. Diabetes may often be intermittent, especially where a rheumatic diathesis exists. In five cases of this kind, Hardy claims to have noticed complete disappearance of diabetes through the employment of the usual dietetic regimen.

The views of the best known authorities as to the prognosis of diabetes are well summed up by P. Bouloumie.[40] Of the

Italian school, Cantani says diabetes is curable on condition that his treatment by beef and lactic acid is instituted not too late and carried out for a long time. Primavera says it is always curable, whatever its stage of intensity. Pietro Berusse had never, up to 1883 seen, a diabetic cured. As to the French school, Bouchardat has been able in a large number of cases to obtain a perfect cure. Andral, out of 84 cases, had the sugar disappear in a very small number, not to return. Martineau has treated 70 cases, obtaining a rapid cure 67 times, and failing 3 times,—a percentage of 95.7 of cure, in diabetics of every age, and presenting the most severe symptoms. In Germany, on the other hand, Seegen, in an experience with 400 cases, has not seen a single cure to such a degree as to permit the use of the carbohydrates in food, such as a well person could take without glycosuria. Frerichs, in his work upon diabetes,—based on 400 observed cases, 282 males and 118 females,—says, "Diabetes terminates in different manner, and not always as unfavorably as is commonly supposed; but unfortunately the termination by cure is very rare." Out of his 400 cases 250 or 62.5 per cent. died, 12 or 3.83 per cent. recovered. The others, it is presumed, had not terminated, but the ratio of cure to death, 12 to 250, was 5 per cent. Lecorché, in his recent work on diabetes in women, says, "If a cure is possible, it is well to remember that the usual termination, after a course more or less variable and a duration more or less prolonged, is death."

Treatment.—In the treatment of diabetes dietetic measures continue to hold the first place.

F. W. Pavy,[41] before the Philadelphia County Medical Society, thought there could be no successful treatment which did not exclude the carbohydrate elements in food. In the case of a patient in middle life, he first puts him on a restricted diet; if the sugar lingers, as is often the case, although diminished, he has recourse, in addition, to opium, codeia, or morphia, believing these substances to have an important controlling influence; in other words, they restore the assimilative power. Certain it is that under the influence of these drugs and a restricted diet, sugar after a time disappears from the urine. After the urine is kept free from sugar a few months, he finds the patient has a certain assimilative power over the starch of bread. He tests this by giving a couple of ounces of bread. If there is no return of sugar in two or three weeks, this is increased to three and then to four and a half, and finally to six. He does not exceed this. Potatoes or starch are never allowed.

The dietetic treatment of diabetes as contrasted with that by drugs has received further support during the past year from the articles of Dr. Austin Flint,[42] who claims that in uncomplicated cases of not more than 12 months standing, careful dietetic treatment, which is always well borne, will produce great improvement, and generally eliminate the sugar and completely restore the general health, although relapses may occur if moderate anti-diabetic diet be not pursued for an indefinite length of time. Dr. Flint is emphatically opposed to the use of milk in diabetes. Against the theory that diabetic coma is the direct result of a restricted diet, he is justly emphatic. In our search for authority in such theory, we have only been able to discover the statement of Lépine,[43] that attention should be called to the danger of suddenly restricting a patient suffering with diabetes to an exclusive meat diet.

The editor of this department,[44] in reply to Dr. Flint's strictures upon the use of milk, calls attention to the fact that it is purely as a dietetic measure that skim milk, exclusively used, will frequently eliminate the sugar more rapidly than any other diet; that it will not do this in every instance; and is not to be regarded as a specific. Unskimmed milk, from some unexplained reason, appears not to have this power.

Seegen relies mainly on diet. He says, however, that while morphine unquestionably exerts a salutary influence on both mild and severe forms, yet its action ceases with withdrawal. Carlsbad water is beneficial only in the mildest form, increasing the tolerance for amylaceous food. The more or less complete exclusion of sugar and starch mitigates the severe form, and causes the sugar to disappear from the urine in the mild form. He has never seen a perfect cure. Cless[45] says the carbohydrates should be excluded whenever possible. Two of his cases recovered so far as to permit the use of large quantities of carbohydrates, five, considerable quantities, and thirteen where the urine remained free from sugar so long as the carbohydrates were excluded from the food. In 29 cases the sugar could not be kept out even by excluding the carbohydrates. Pavy[46] calls attention to the unsatisfactory character of some of the articles of food sold for the special use of those suffering with diabetes. The chief difficulty is in the use of the substitute for bread. Gluten flour and gluten bread always contain a certain amount of starch, and may be considered in good state if this does not amount to more than 25 or 30 per cent. The actual figures in two samples of gluten bread from the same source in Pavy's hands were 74 and 76 per cent., while two samples of gluten flour from the same establishment contained 80 and 82 per cent. A biscuit made in France, purporting to be made of gluten flour, contained 84 and another 89 per cent. of starch. An analysis of Hecker's gluten bread, made a short time ago by a reliable chemist in Philadelphia, discovered 76.35 of starch, another of the Health Food Company's bread discovered 65.45 of starch, while Vienna white bread contained but 85 per cent. A more recent analysis made in Boston of the Health Food Company's diabetic flour, discovered 75 per cent. of sugar-forming matter.

Dr. Pavy also investigated bran biscuits. The figures yielded by some were 38 per cent. of carbohydrates expressed as starch, while in others they amounted to 50 and 60 per cent., and even upward. Besides starch, bran contains lignose and cellulose. These, when boiled in sulphuric acid in the process of analysis, are like starch, converted into glucose, and thus contribute to the result obtained; but as they are hardly likely to be affected by the digestive fluids, they escape conversion into glucose during digestion. The starch obtained from bran by ordinary boiling water will amount to 23 per cent. In the case of the bran prepared for the use of the diabetic, it amounted to about the same, going to show that it had received no special treatment. None of the samples of macaroni, vermicelli or semola could be regarded as suitable, analysis giving a range of from 75 to 81 per cent. of starch, while the ordinary macaroni yields 86 and vermicelli 87 per cent.

Coignard,[47] of Paris, says he has empirically advised, for many years the substitution of potatoes, either simply boiled, or better still, *en purée*, for the gluten bread of commerce in the diet of diabetics, before he was aware that the tubers contained less sugar-producing substance. He gives a table showing the amount of sugar-forming material in each of the substances named, estimated by actual conversion into sugar by prolonged boiling in acidulated water, and then determining the quantity by trituration with Fehling's solution and the polaroscope. The analysis was made by five different chemists with the following results: Potatoes boiled in water, 17.14; purée of ditto, 8.32; fried, 33.30; gluten bread No. 1, 31.60; gluten bread No. 2, 31.15; gluten bread No. 3, 32.00; gluten bread No. 4, 62.50; ordinary bread, stale, 60.00; ordinary bread, new, 51.00; crumb of bread, 54.71; crust of bread, 76.14; crust of bread, toasted, 84.15; biscuits of fine flour, 70.94; biscuits of fine flour, high dried, 88.89. The absurdity of permitting the crust of wheaten bread to be eaten, while the crumb is forbidden, is pointed out and confirmed by an examination of the table. He adds that baked potatoes, although giving more starch than an equal weight of boiled potatoes, still contain less than the best gluten bread. In frying, the loss of water raises the proportion of starch. When the extremely erroneous ideas formerly prevalent as to the freedom of gluten bread from starch, are considered, it is not surprising that the use of potatoes should not place them at a relative disadvantage with bread. At the same time, we caution our readers against adopting Coignard's views until they have been confirmed by others.

A. Pollatschek,[48] of Carlsbad, unites with Aducco and Mosso of Turin, and Salkowski, of Berlin, in claiming that saccharin or anhydro-ortho-suphamide benzoic acid is harmless in diabetes, and may be given for sweetening purposes, neutralized, as proposed by Leyden, by sodium carbonate. Fischer,[49] of Berlin, suggests the following: saccharin, 3 grams; natri carbon. sicci, 2 grams; manniti, 50 grams. Make 100 pastilles. One of these may be used to sweeten a cup of coffee, tea or cocoa. According to Saundby, a convenient solution of saccharin may be made by using bicarbonate of sodium as a solvent in the proportion of 5 grams and 10 grams of saccharin to one ounce of water. A teaspoonful is sufficient to sweeten a cup of coffee. Saccharin may be used advantageously in the following combinations: sodii acet., ʒiiss.; aquæ carbonat., ʒxxiiss.; saccharini, gr. iss.; essent. citr., ♏xl.; which may be taken three or four times daily. Also, sodii tartrat., ʒviiss.; aquæ carbonat., ʒl–lxxv.; saccharini, gr. v.; essent. citri, ♏lxxv. To be taken several times daily, as occasion demands. Saccharin is said to be 300 times sweeter than glucose, antifermentative, and is said to have antiseptic properties.

Jaccoud,[50] in his dietary for diabetics, not only omits all sugars, including sugar of milk, glycerine, and milk itself, but also fats, including eggs, not because of the white, but because of the yellow, which he thinks has an influence on the formation of sugar. The non-starchy vegetables he allows.

It has generally been conceded that where sweetening of some kind is insisted upon by diabetics, glycerine is admissible for this purpose. Pavy and others some time ago claimed that glycosuria increased under its use. Later, Weiss and Luchsinger claimed to have shown that under its use glycogen accumulated in the liver. Still later, Luchsinger and Eckhard ascertained that glycosuria experimentally induced could sometimes be prevented by the subcutaneous injection of glycerine.

W. B. Ransom[51] recently repeated these experiments, formulating the following conclusions: (1) That certain forms of glycosuria may be checked by glycerine. (2) That glycerine acts more efficiently when introduced by the alimentary canal than when injected subcutaneously. (3) That glycerine checks glycosuria by inhibiting the formation of sugar in the liver. (4) That in this way glycerine may lead indirectly to an accumulation of sugar in the liver.

Ransom holds that the formation of glycogen in the liver is due to cell metabolism and not to the action of a ferment, and that glycerine acts by modifying the protoplasm of the liver cells. He has no opinion as to the therapeutic value of glycerine in diabetes. The views of clinicians are various and conflicting, while different results may follow different doses and modes of administration.

In seeking to apply these results to the treatment of diabetes, it would seem that glycerine ought to be useful in the variety in which glycosuria is the result of a too rapid production and discharge of glucose from the liver rather than in alimentary glycosuria, where the absorbed glucose of intestinal digestion is carried through the liver too rapidly or in too large quantity to permit its conversion into glycogen and storage in the liver. It is not impossible, however, that a careful further clinical study of the effects of glycerine may lead to more positive conclusions as to its utility in diabetes.

Medicinal Treatment.—Perhaps no treatment suggested for diabetes has ever attracted more attention than that published by Martineau,[52] and suggested to him by Rouget. The directions are as follow: Add to a litre (one quart) of water, in the upper globe of a Briette apparatus for making aerated water, 20 centigrams (3 grains) of lithium carbonate, and a tablespoonful of the following solution: distilled water 500 grams (1 pint), arsenite of sodium 20 centigrams (3 grains). This quantity should be drunk during meals, mixed with wine, and the apparatusful should last for at least three meals. Between meals, if the patient is thirsty, he should drink nothing but this water. The diet need be modified only so far as to limit the use of starches, fruit and sugar. Martineau adopted this treatment in 1875, since when he has collected 70 cases, in 67 of which he made a "rapid cure," in 3 he failed. The 67 cases were of diabetes occurring in arthritic patients, the condition manifesting itself either in the arthritic form, or in the shape of biliary or urinary lithiasis, or as cutaneous eruptions. In the three in which he failed, the arthritis was wanting, "but herpetic symptoms existed under the form of 'nervovism.'" He considers lithia the efficient factor in this treatment, since all the mineral waters which have a reputation for the cure of diabetes contain lithia. He suggests that it conspires with the bicarbonate of sodium to transform the glycogenic matter of the tissues first into glucose, and then into carbonic acid and water; that associated with arsenic, it increases the number of red blood corpuscles, and renders combustion more active; it stimulates the nerves of organic life, upon whose influences the processes of nutrition depend, and that it remedies the sluggishness of the nutritive processes. In lieu of the solution of the carbonate of lithium and arseniate of sodium recommended by Martineau, Vigier[53] gives the following formula for a pill containing the same ingredients: ℞ lithii carbonat., gr. $1\frac{1}{2}$; sodii arseniat., gr. $\frac{1}{20}$; extr. gentian, gr. $\frac{1}{4}$; for each pill. One pill should be taken morning and evening. The convenience of a pill as compared with solution, especially to persons traveling, is manifest; and as the carbonated water can only be regarded as an accessory in quenching thirst, the pills should be quite as efficacious. Créquy, Vigier, Delpech, C. Paul and Dujardin-Beaumetz reported more or less good results from the use of the arsenical lithiated treatment.

Le Blond[54] advises the administration of an aereated water made in Paris for the past few years, which differs from the familiar water in that it is gaseous with oxygen instead of carbonic acid. Three cases are reported in which the treatment caused the total disappearance of sugar from the urine. In a fourth, partial failure was said to be due to imperfect oxidation from chronic bronchial catarrh. This may be regarded as a revival of the treatment by oxygen formerly suggested in the shape of peroxide of hydrogen. Duchenne[55] recommends the following mixture for the thirst of diabetes: phosphate of potassium, 2 parts; water, 75 parts. One teaspoonful should be given three times a day in a little wine or hot tea.

Further observations on the use of morphine in diabetes have been made by Mitchell Bruce,[56] in which he observed an almost total disappearance of sugar from the urine when associated with a restricted diet. The drug was introduced by the mouth and rapidly increased, so that on the ninty-ninth day 7 grains of acetate of morphia were given. When, however, the drug was used hypodermically, one-fourth of a grain thrice daily, increased one-twelfth every three or four days, until a maximum of three grains a day were given, drowsiness necessitated a diminution of the amount. At no time during a period of 49 days were less than 760 grains of sugar secreted daily. The amount excreted on a rigid diet without drugs, ranged from 1360 to 4000 grains a day. Whence Bruce concludes: (1) that the drug had much less influence when introduced by the general circulation than when absorbed by the portal circulation; (2) the increased income of sugar was not from the intestine or portal vein, for when all of saccharine or amylaceous articles of food are eliminated, the patient continues to excrete large quantities of sugar; (3) the excessive glycosuria was effected mainly or entirely in the liver, and not in the other viscera or muscles, because (*a*) when the morphia was introduced into the liver by the portal vein, the secretion of sugar was nil, while it did not materially affect the other viscera, such as the central nervous system; (*b*) when it was introduced into the general circulation only to the degree that might have been expected from the portion that would reach the liver through the hepatic artery, its effect was much less. (4) These results appear to prove that in this instance, if the diabetes was of nervous origin, the seat of the disordered process—he does not say the seat of the nervous derangement originating it—was in the liver and not in the central nervous system or nerve trunks. This followed also from the fact that morphia under the skin profoundly depressed the nervous system, producing drowsiness, while it did not affect the glycosuria.

Villemin[57] reports a well-marked case of diabetes treated by belladonna and opium combined. On the 10th of March he gave one-tenth of a gram of extract of belladonna, and five-hundredths of a gram of opium daily. By the 27th the urine had decreased from 12 and 14 litres, and the sugar from 58 grams per litre to 40 grams. The dose of the extract was gradually increased until on the 15th of May .15 of a gram of each were given daily, by which time the urine had decreased to 3 and 4 litres, and the sugar to 2 and 5 grams per litre. On the 20th, the opium and belladonna were each increased to .20 of a gram, and eight days later there was only a trace of sugar. For 17 days the treatment was continued without any reappearance of the sugar. On the 14th of June the treatment was suddenly discontinued, and in two or three days the glucose increased to 16 grams. On the 17th the dose of .2 of a gram was restored, and by the 22d the sugar had again disappeared, and the urine was reduced to 2 litres. Up to the 20th of July a diabetic diet was used, but after that time a diet including potatoes, bread and macaroni was permitted, without any return of the sugar, while the dose continued at .2 of a gram. From the 28th of August to the 12th of September bromide of potassium was substituted, with a rapid return of sugar to 45 grams and an increase of sugar at 11.5 litres.

With a view to deciding whether it was the combination which was beneficial, one or the other of the drugs was suspended; and in either event the glucose and quantity of urine increased. The beneficial effect was not merely upon the urine, but the other symptoms of diabetes disappeared, and the patient's general health improved simultaneously, he gaining 8 kilograms in weight while under treatment. Further observations were prevented by the patient leaving the hospital.

Robert Saundby,[58] upon the results of treatment of many cases with and without opium, declares that in opium we possess the most valuable and only trustworthy drug for the treatment of this disease. Quinine he regards as comparatively inert, even in much larger doses than are suggested by its principal advocates. Opium is especially valuable for its influence in diminishing the amount of urine, an opium pill often giving an undisturbed night to patients who otherwise have to rise frequently. Salicylate of sodium, he says, may be of some service as an alkali, for he is strongly in favor of the alkaline treatment; but he has never observed it to produce any specific effect upon the quantity of urine or sugar. Arsenite of bromine, whether used in its pure state or in the form of Clemens' solution, has never in his hands justified the praise it has received from some, after an experience of six cases with its use. Bromide of potassium is, in his opinion, the best routine remedy to employ in conjunction with opium, although it does not deserve the name of a specific. This drug also failed in the hands of Villemin,[57] who gave it in doses gradually increased from 4 to 12 grams daily without effect. Saundby calls attention to Mr. Lawson Tait's success with sulphuret of potassium ointment in the treatment of the pruritus vulvæ, and has himself been very successful with an ointment of borax.

Pepsin has been used with satisfaction in the treatment of diabetes mellitus by the late Surgeon-Major E. B. Gardner,[59] of the British Army. He began its use 25 years ago. The case reported was that of a native of India who had had the disease for about two years, being blind also with cataract. He was restricted to milk or buttermilk, bran bread, and meat two or three times a week. The urine immediately diminished in quantity and became of lower specific gravity, and at the end of six weeks, the man was so far improved, though not well, that he was able to take starchy and saccharine matters with impunity. The only other treatment was an occasional dose of castor oil and pure distilled water colored with permanganate of potash, in order to satisfy the patient that he was taking enough medicine. The advantages claimed for the treatment are (1) that it can be given in addition to other remedies and with due observance of dietetic rules; (2) that it admits of a relaxation of rigid diet before it becomes injurious; (3) that it never causes, but frequently relieves any concurrent affection of the skin.

Salicath[60] has used with advantage the preparations of pepsin with the total disappearance of sugar. He believes that the pepsin changes the grape sugar into lactic acid.

J. M. DaCosta[61] continues the use of salicylate of sodium in diabetes in doses of a drachm and a drachm and a half in 24 hours. He is said to claim better results with this than with any other remedy. J. C. Wilson [62] favors the combined use of sodium salicylate and Fowler's solution in the dose of 15 grains of the former and 5 drops of the latter 4 times daily. He obtains from the combined use results not obtainable from either alone. The editor's experience in a single case goes to confirm Dr. Wilson's conclusions.

George C. Kingsbury[63] has treated a case of diabetes with apparent good results with jambul seeds (not jumble beads or jequirity seeds). The patient had been ill for over six months, was greatly emaciated and very much prostrated. He had great thirst and a ravenous appetite. The urine had a specific gravity of 1042, and from 7 to 7½ pints were passed in 24 hours. 5 grains were given 6 times in 24 hours for a fortnight, at the end of which time the patient was able to get up, was able to walk out for an hour at a time, was neither thirsty nor abnormally hungry, was passing 4 or 5 quarts of urine with a specific gravity of 1020. The diet was not restricted in any way. E. Harry Fenwick[64] reports a diminution of sugar and urine and rapid healing of sloughing ulcers in diabetics. No mention of number of cases is made, but one striking one is related,—under the use of jambul, 2½ gr. to 5 gr. in pearls thrice daily after food. Robert Saundby[65] used jambul in 5 grain powders 4 and 3 times daily in 5 cases of diabetes. No case was cured, but all left the hospital more or less relieved by treatment. All were carefully dieted, and besides the routine diet they were allowed to drink potash imperial sweetened with glycerine, and in several instances were treated with vapor baths. Uniformity of conditions were observed during treatment and its omission. There were eight distinct trials in the 5 cases, 5 being followed by an increase and 3 by a decrease of sugar. Its disuse was followed in 4 cases by a decrease and in 2 cases by an increase.

E. K. Weller[66] has used cocaine in the case of a farmer's wife aged 54, with a nervous and excitable temperament manifesting itself in a quarrelsome disposition, which made her married life one of continual strife, her husband having a similar temperament. She developed typical symptoms of diabetes, including thirst, the passage of a large amount of saccharine urine, dryness of the skin and mucous membranes, and a decided anæmia. Weller prescribed 2 drops of a 4 per cent. solution of cocaine every 3 hours, together with an anti-diabetic diet. In a few days the polydipsia disappeared and the urine was little above the normal. An annoying pruritus vulvæ diminished, as did the itching and dryness of the skin. He continued the treatment, adding 2 drops of tincture of opium and 18 of tincture of the chloride of iron 3 times a day after meals. Three weeks later all the symptoms had disappeared, and she felt "as well as ever." There was no return of symptoms two months later. The fact that an anti-diabetic diet was associated with the treatment makes it impossible to ascribe the improvement unqualifiedly to the cocaine. Further observations are therefore necessary before it can be accredited with therapeutic value. Weller's idea is that it acted as a sedative upon the emotional centres in the neighborhood of the sugar-forming centres in the medulla.

Bufalini,[67] of Siena, found thymol, in two cases, most efficient in warding off the gastro-intestinal symptoms, anorexia, vomiting, borborygmi and diarrhœa, which so often occur when an albuminous diet is persevered in, and which are often followed by a reappearance and steady increase in the sugar. He also found it of service as an intestinal disinfectant. The two patients upon whom he experimented both suffered from acetonæmia, the breath exhaling the odor of acetone. This was caused to disappear by the use of thymol, and the general condition improved.

Joseph Levi, Correspondent of the ANNUAL in the Virgin Islands, reports 4 cases treated by iodoform in pills of $\frac{1}{6}$ grain, 3 times a day, there being a reduction in the quantity of urine and percentage of sugar. Carbohydrates were omitted from the food, massage was used night and morning and apparently did much good. The patients were 3 males, aged 40, 54 and 19 years respectively, and one female, aged 34. In 3 cases furuncles made their appearance, but began to dry up after the iodoform was taken regularly for 3 weeks. For a time the patients lost in weight, but eventually gained again.

Goenner[91] has recently tried antipyrin, in daily doses of 3 grams, in a case of diabetes of about seven years' standing. The patient was a gentleman, aged 60, with 2.8 per cent. of sugar in his urine. In three days the urine gave but an indistinct

Trommer's reaction. Not a trace of sugar remained after the patient had taken 27 grams of the drug.

For the treatment of the genital eczema of diabetes, Fournier[68] recommends, in addition to the general treatment for diabetes, baths, at first moderately alkaline, afterward highly alkaline, frequent alkaline lotions, injections of the same composition, and finally the use of drying powders.

The deafness accompanying diabetes, according to Miot, is best treated by the continuous current, which diminishes the congestion of the mucosa and allows the introduction of bougies and galvano-caustic agencies.

Cornillon[69] has recently employed subcutaneous injections of ergotin to diminish the thirst of diabetes, with the most successful results, succeeding when the most rigorous dietetic measures failed. A daily injection of from 5 to 6 drops answered the purpose, and was followed by no bad results.

Treatment of Diabetic Coma.—Stadelmann having shown that a very considerable amount of β oxybutyric acid is found in the urine in diabetic coma, has suggested that the dyscrasic condition thus produced be counteracted by large doses of sodium carbonate, administered by the mouth, by enema, or by hypodermic injection. Acting upon this, Lépine[70] reported a trial. There was a strong acetone odor in the breath, but no coma as yet, when he attempted to administer a large dose by the mouth, but failed because of the objection of the patient to this mode of administration. The patient soon fell into coma. He then threw into the median cephalic vein, of sodium bicarbonate 34 grams, sodium chloride 8 grams, and water 1.5 litres. Encouraged by a slight amelioration of the symptoms, he repeated the injection, using 10 grams of sodium chloride, and only 10 of sodium bicarbonate. Notwithstanding the report of the nurse in attendance that the patient was much better in the evening, he died the next morning. Lépine nevertheless commends the alkaline treatment, not so much because of the neutralization of free acid in the blood, since mere neutralization will not annul the toxic action of the acid, but because alkaline injection may assist in breaking up the acids and favor their elimination.

In the case related he found that β oxybutyric acid disappeared from the urine after the injection. Prior to the injection, both the blood and urine yielded this acid in parallel amount. He admits that the better way would be to prevent the formation of the acid, but such method has not yet been discovered.

Jaccoud[71] recommends for the treatment of diabetic coma alkaline purgatives and inhalations of oxygen, but says that when once the stage of coma is reached, treatment is hopeless.

DIABETES INSIPIDUS.

Etiology.—Adolfo Berri[72] reports a case of persistent polyuria after fracture of the base of the skull in a man of 36, the fracture being caused by a fall. There was also thirst, both symptoms coming on a fortnight after the accident. The maximum quantity of urine passed was 140 ounces, and there was neither albumin nor sugar. The polyuria ceased a month after, and ten weeks later the man died of phthisis. At the autopsy, traces of the old fracture were found, and the remains of a hæmorrhage in the floor of the fourth ventricle. Berri collected thirteen other cases of the same kind. Three terminated fatally. No lesion of the floor of the fourth ventricle, pons or medulla was found in any of them, but all the conditions were such as would lead to pressure in this vicinity. The average duration of the polyuria was a month or six weeks, and the quantity of urine varied greatly, reaching, in one instance, 30 litres or 52½ pints. In a few cases, sugar was slightly present at first, but in almost all disappeared before the polyuria. Kahler[73] collected 22 cases of simple persistent polyuria, associated with coarse cerebral disease. Of 14 autopsies, tumors, mostly of the base, were found in 10. Of the remaining 4, one showed atrophy of the right half of the medulla, another superficial congestion along the floor of the fourth ventricle, a third softening in the same area, and the fourth an apoplectic clot in the medulla. More than 30 years ago, Bernard showed that puncture of the floor of the fourth ventricle produced simple polyuria, glycosuria, or both, according to the exact site of the puncture. This condition, however, lasted but twenty-four hours. Bernard's experiments were repeated by Eckhard and Kahler. The latter produced localized destruction of tissue by means of nitrate of silver solution; and persistent polyuria by irritating in this way various areas in the lateral part of the medulla and pons. In fracture of the base of the skull the result is probably due to pressure brought about by blood effusion, and in brain tumors by pressure of the new growths. The absence of appreciable lesion in these localities in other forms of diabetes mellitus and insipidus, make it impossible for us to locate the lesions of either diabetes insipidus or diabetes mellitus solely in these centres, or at least until our methods of research enable us to discover changes not appreciable with our present facilities.

Kraus[74] reported to the Society of German Physicians in Prague, the case of an epileptic who had diabetes insipidus. He, with a healthy man as a control case, was made the subject of experiment by Kohler, in whose clinic the case occurred. When large amounts of fluid were ingested by both, the diabetic urinated quickly as compared with the sound man. When large quantities were continuously given, the diabetic was relatively polyuric, but only when thirst supervened secondarily did he become absolutely polyuric.

Treatment.—Demange[75] says that diabetes insipidus is best treated by valerian in doses of two to four grams of the powder per day. Smith Seaforth[76] treated a typical case with 30 minim doses of the fluid extract of ergot 3 times a day; also by a pill containing 1½ grains of extract of belladonna, with half a grain of opium daily, to no purpose; but under the use of Clemens' solution of bromide of arsenic after each meal, and dilute phosphoric acid in small doses to assuage thirst, the quantity of urine fell from 14 pints to 5 pints daily, and the specific gravity rose from 1003 and 1005 to 1012. The patient's general health also improved, but the polyuria continued in the reduced degree. C. E. Todd[77] reported to the Adelaide Medical Society a case of diabetes insipidus treated with codeine with beneficial result, but no note of the dose is given.

DISEASES OF THE SUPRARENAL CAPSULES—ADDISON'S DISEASE.

Morbid Anatomy.—The majority of observers claim that the essential lesion of Addison's disease is caseous tubercle of the suprarenal capsules, with more or less atrophic disturbance of these bodies.

In the literature of the past twelve months, the following case reported by E. Belaieff,[78] of Toronoj, is alone opposed to this idea. It is further interesting in consequence of the extreme youth of the subject, no case younger than three years having been previously reported. A male child, apparently seven days old, was found in the street. The integuments were slightly yellowish, with a gray tint. Later, the yellowish discolorations disappeared, and the skin gradually assumed a dirty gray color, most intense on the back and belly, and especially about the navel. Notwithstanding a good appetite, the child gradually became very weak and emaciated, and died 53 days after admission to hospital, having become unconscious during a paroxysm of tonic and clonic convulsions. There was no fever. The autopsy discovered that both suprarenal capsules were considerably enlarged, measuring 1 in. by 3-4 in., their upper two-thirds being transformed into an aggregate of thin-walled, semi-transparent cysts, varying in size from a pin's head to a small cherry. Their contents were a clear serous fluid.

The following cases, nine in number, all go to sustain the prevalent view, that Addison's disease is exclusively due to caseous tuberculosis of the suprarenal capsules. Goldenbaum,[79] of the Pathological Institute of Dorpat, records a case in which bacilli were found, by Ehrlich's method, in the suprarenal capsules of a genuine and typical case of Addison's disease. He calls attention to the fact that P. Guttmann[80] and Rauschenbach[81] had recorded cases where bacilli were found in the capsules, but in addition many other organs were caseous, and in neither of these cases were any symptoms of Addison's disease present. In Goldenbaum's case, there was no caseation in any other part of the body, and bronzing of the skin and mucous membrane was well marked. Barron[82] found the bacillus of tubercle in the caseous matter of the suprarenal capsules in two cases of Addison's disease. H. Dauchez[83] reports a case of Addison's disease in a man of 40, in which the characteristic symptoms were present, discolorations, profound adynamia without appreciable cause, and total anorexia. At the autopsy, both suprarenal capsules were found enlarged to twice the normal size, irregularly mammillated, and non-adherent. Section revealed cheesy nodules about the size of a grain of maize, full of pus, and each surrounded by a thickened, fibrous envelope. The disease was regarded as primary miliary tuberculosis. The left capsule was 7 centimetres long, the right 6, the height of the capsules 6 centimetres. The other organs were normal except a cheesy, submaxillary lymphatic gland. Courteen[84] reports a case of typical Addison's disease in a boy of 16, accompanied by the characteristic bronzing and asthenia, in which the autopsy revealed the right suprarenal capsule much enlarged, weighing one ounce and consisting of a soft, pale yellow, cream-cheese like substance. The left capsule was still larger, weighed 1¼ oz., and was of the same substance as right. No microscopical examination was made. Other organs normal, except the spleen, which was of a pulpy, grumous consistency, and weighed 10 oz. No disease of vertebræ. In a typical case of Addison's disease under F. K. Owen,[85] the adrenals were greatly enlarged and full of concretions and small caseous nodules. There were also small deposits of cheesy matter and little pockets of pus in the kidneys. Davidson[86] had a case of Addison's disease dying at 33 of exhaustion three months after the appearance of the symptoms. There was great muscular weakness, vomiting, and abdominal pain and bronzing. The suprarenal capsules were masses of curdy, tuberculous matter, and were matted to the surrounding tissues. The left testicle was tuberculous, and there was an encysted empyema in the right pleura. Harrington Sainsbury[86] showed to the Pathological Society of London the adrenals from a case of Addison's disease. The case was interesting because of a traumatic history, and because one organ was much more advanced in atrophy than the other, and threw light on some cases recently described as occurring in association with atrophy of the capsules. They illustrated the degree to which, subsequent to fibro-caseous change, atrophy may proceed, and suggested that some of the cases described as Addison's disease with simple atrophy, might really be examples of ultimate stages of the fibro-caseous change. It also suggested that some of the cases recorded absence of the capsules in association with symptoms of Addison's disease might be examples of extreme atrophy and practical effacement of the organ. Samuel Wilkes said that he had always insisted that the atrophy of the organs was the main point of the disease, and that no case had ever been presented proving that an adventitious growth of the adrenals could cause characteristic symptoms of Addison's disease. W. B. Haden said, also, that the essential point was destruction of the adrenal bodies, but that the nature of the destroying process was of secondary consequence. D. S. Lamb[87] presented to the Medical Society of the District of Columbia the suprarenal capsules from a woman of 31 who, for three years before her death, had presented gradually increasing bronzing of the skin to which was added, some months before death, the extreme debility characteristic of the disease. The suprarenal capsules contained cheesy masses, larger in the right. There were calcareous deposits in the lungs and in the omentum.

It is plain, however, from the above, that the view commonly held that Addison's disease has for its anatomical lesion, tuberculous infiltration of the suprarenal capsules, for the most part in a state of advanced cheesy change, is sustained by the morbid anatomy of the cases reported.

Diseases of the Capsules Unaccompanied by Bronzing of the Skin.—The following case illustrates the occasional presence of caseous matter without bronzing of the skin. Davidson[88] reported a case in which the structure of the suprarenal capsules was replaced by caseous and calcareous matter. The right was enlarged, hard and fibrous, with small calcareous deposits. Both were adherent to adjacent tissues. There was extreme exhaustion and emaciation, but no bronzing of the skin. Toupet[89] presented to the Anatomical Society of Paris, two hypertrophied suprarenal capsules containing hæmorrhagic cysts. The patient died of uræmia, and did not present abnormal pigmentation. Microscopic examination of sections showed the disappearance of tubular substance, with certain vesicles dilated and the walls in a state of fatty degeneration. There was interstitial hæmorrhage. Analogous cases are reported.

Lamb[89] presented to the Medical Society of the District of Columbia two specimens of cancer of the suprarenal capsules, both from women, neither of whom had any bronzing of the skin. One case, a woman of 61, was a case of general carcinomatosis primary, probably, in the stomach. The second, a woman of 35, had cancer of the right suprarenal capsule.

Richard May[90] has studied these organs from 42 necropsies other than cases of Addison's disease, and discovered ten accessory capsules, eight being composed entirely of cortical substance, and two of cortical and medullary. They lay in one case under the capsule of the organ, in others in the fatty substance surrounding it, or close to the solar plexus or in the kidneys, forming the so-called *strumæ lipomatodes aberratæ renis;* others lay in the broad ligament. Pathological changes were found in thirty-eight out of the forty-two necropsies. Most frequent was cloudy swelling in cases of scarlatina, erysipelas, sepsis after the radical operation for hydrocele, croupous pneumonia, purulent pleural effusion and peritonitis,—in short, in those cases where infection from pathogenic micro-organisms existed. In advanced forms of this condition, infiltration, with small cells or even suppuration, was noted. Fatty degeneration of the capsules was also very common, either as the result of cloudy swelling, or as a primary state caused by defective vascular supply in cases of anæmia and diseases of the respiratory and circulatory organs. Engorgement of the capillaries was discovered in subjects who died of long-standing heart disease. In two cases, tubercular caseation of the capsules was found. It is to be remembered that in none of these cases was there bronzed skin, but in both there was pulmonary tuberculosis, and in one tubercular basilar meningitis. In one patient, who died of primary cancer of the breast, one suprarenal capsule was found converted into "fibro-carcinoma."

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DISEASES OF THE KIDNEYS AND BLADDER.

BY FRANCIS DELAFIELD, M.D.,

NEW YORK.

BRIGHT'S DISEASE.

Etiology.—The theory that inflammation of the kidneys is due to the irritation of these organs by morbid substances, which pass through them in the performance of their natural functions, is one that is still popular with the profession. A very plain statement of this theory is made by Dr. Thomas:—[1]

"I believe that the contracted kidney is always caused by the presence in the blood of some material which has to be daily excreted in large quantities, and which, in its passage through the kidneys, gives rise to an increased formation of the fibrous stroma and other subsequent changes."

We find that alcohol tends to produce it, and we know that in such patients, from chemical examinations of the urine, the kidneys have to excrete a goodly portion of what has been taken in.

We often find that patients who have to do a large amount of mental work, or who have had a good deal of mental worry, are apt in time to suffer from a contracted kidney. The writer has noticed such patients for years, and invariably found that they have passed a large quantity of phosphates daily. These patients do the bulk of their mental work in the morning, and the effete material the kidneys have to excrete is passed somewhat later. He has further observed that later on, after excessive work or very great worry, the phosphates are accompanied by albumen.

In gout there is a tendency to the formation of urates and uric acid in the blood. The kidneys for years have excessive work to perform in excreting these substances, and the writer believes that in consequence of this, a granular state of the kidneys is induced.

In plumbism we find that the patients pass urine containing lead in solution daily, and after this has continued for a time albumen begins to appear.

Many patients, especially those who are dyspeptic, pass large quantities of oxalates daily. Such patients, later on, pass albumen also, and ultimately have Bright's disease.

The conclusion the writer has come to with regard to the causation of the contracted kidney is this: that it is invariably preceded by the presence of large quantities of some material in excess, such as phosphates, urates, oxalates, lead, alcohol in the urine; that after a time a small quantity of albumen will be found accompanying it; that the presence of these constituents constantly in the blood, present there for excretion, gives rise to the hyperplasia of fibrous tissue and subsequent changes." Such mechanical views of the causation of nephritis will probably continue to prevail until physicians and pathologists learn to compare the lesions of the kidney with those of the lungs, the heart, the arteries, the stomach, the liver and the spleen.

The views of Semmola[2] concerning the nature of Bright's disease, although they are not likely to be accepted without considerable modifications, are yet well worthy of consideration:—

"True Bright's disease consists of a chronic morbid state distinguished by: (1) Its etiology, the slow action of damp cold on the skin. (2) By impairment leading to entire abolition of the functions of the skin, depending on the progressive ischæmia with atrophy of the sudoriparous glands, progressive atrophy of the layer of Malpighi, and proliferation of the connective tissue of the derma. (3) By a chemico-molecular alteration of the albuminoids of the food; distinct alteration of their diffusibility, and hence inassimilability and necessary elimination through all the emunctories, and chiefly by the kidneys. (4) By progressive diminution of the combustion of the albuminoids, which shows itself by progressive diminution in the formation of urea, so that a progressive diminution results in the quantity of urea eliminated by the urine in the 24 hours, without any accumulation of this principle in the blood or elsewhere. The blood in Bright's disease (in patients not in the uræmic condition) contains less urea than that found normally. (5) By a subcutaneous serous infiltration, surely and slowly progressive and having no relation to the hydræmia. (6) By a special cachexia not in relation to the losses of albumen, but to general impairment of assimilation. (7) By a secondary development of an inflammatory process of both kidneys.

Lesions.—The changes which take place in the glomeruli in acute nephritis have for several years attracted much attention and have been described by Langhans,[3] Friedländer,[4] Nauwerck,[5] and others. Such changes in the glomeruli, it is said, may exist by themselves, and then we may speak of a "glomerulo-nephritis;" or they may be associated with other lesions of the kidney. In the slighter degrees of inflammation there is only a swelling of the endothelial cells of the capillaries. In more severe degrees the endothelium is much swollen, the glomerular and the capsular epithelium are also swollen, and there is coagulated matter in the cavity of the capsule.

The growth of endothelium may be so great that the capillaries are filled with endothelial cells and white blood cells until they are imperviable for the blood current. In other cases masses of white blood cells collect in the capillaries or hyaline thrombi are formed in them; or the hyaline membrane, which forms the wall of the capillaries outside of the endothelium is swollen; or there is a formation of hyaline matter between this membrane and the endothelium, and by both these changes the capillaries are obliterated. These views have met with very general acceptance and have been adopted by some of the text-books. Recently Hansemann,[6] after a study of 120 cases of nephritis, has arrived at somewhat different results. He believes that in the majority of cases the nephritis does not begin in the Malpighian bodies, although changes in these bodies are associated with nearly all examples of acute nephritis; that the principal changes in the Malpighian bodies are a proliferation of the epithelium of the tufts and an accumulation of leucocytes; and that it is a mistake to attach too much importance to the lesions of the glomeruli and overlook the other changes in the kidney.

Some progress has been made in appreciating the real nature of the waxy or amyloid kidneys, and in recognizing that waxy degeneration of the blood-vessels may exist either with or without other lesions of the kidney. Litten[7] has reported 11 cases of waxy degeneration of the glomeruli, without other lesions in the kidneys and without albuminuria. He also calls attention to the close relationship that exists between hyaline and amyloid degeneration, and the probability that these two bodies represent different stages of intimately related processes.

Attention has been called by Holt[8] to the frequency with which acute nephritis occurs in young children as a primary disease. He has collected 23 cases of this kind, partly from his own practice and partly from that of others. In all of the cases, cerebral symptoms were marked, there was a decided febrile movement in 10, while dropsy was present only in 5. The urine was scanty in some, abundant in others, albumen and casts were regularly present, but not always early in the disease. It is evident that such a primary nephritis in children, with cerebral symptoms and without dropsy, may readily be mistaken for meningitis.

Concerning the lesions of puerperal nephritis, we find a very good paper by Weinbaum[9] reviewing the literature and adding three cases of his own.

Frerichs considered puerperal albuminuria to be due to an acute nephritis, and that the obstacles to the circulation of blood in the abdomen, with changes in the composition of the blood—diminution of the red cells, and increase of the white cells and of the fibrine—are the causes of such a nephritis. He recognized the fact that, although the disease is usually temporary, it may become chronic.

On the other hand, Rosenstein in the first two editions of his work on the kidneys held that in these cases there was no active nephritis at all, but only a chronic congestion due to the pressure of the pregnant uterus on the veins.

Bartels, again, went back to the teachings of Frerichs and described the "acute parenchymatous nephritis of pregnancy," but acknowledged his inability to find a cause for the nephritis. Leyden proposed an entirely different view of the subject. He considered the condition to be neither one of acute inflammation, nor of chronic congestion, but rather one of arterial anæmia, and that the changes in the kidneys are those of degeneration. But he also recognized the fact that some cases of puerperal kidney disease pass on the chronic form and resemble ordinary cases of Bright's disease. The disease, according to Leyden, is regularly developed in the second half of pregnancy, most frequently in primipara.

The intensity of the disease, especially of the albuminuria, increases toward the end of pregnancy, is most marked during parturition, and rapidly decreases after this.

Albuminuria without dropsy is much less common in pregnant women than is dropsy without albuminuria.

The cases related by Weinbaum seem to be examples of acute diffuse nephritis passing on to the chronic stage.

Apparently the renal conditions which we have to recognize during the latter months of pregnancy are these:—

(1) Dropsy without any kidney lesions.

(2) Swelling and degeneration of the renal epithelium,—a transitory condition.

(3) Acute exudative nephritis, with the exudation of blood serum and consequently large quantities of albumen in the urine, is a more severe disease, but also a temporary one.

(4) Acute diffuse nephritis, which involves the stroma of the kidney, and not infrequently becomes chronic.

Letzerich[10] describes a form of nephritis, of which he has seen 25 cases, under the name of "nephritis bacillosa interstitialis primaria." All but 3 of the cases were in children between the ages of 1½ and 13 years, the remaining 3 cases were in young women aged 19, 23, and 20 years. There were only four deaths, all of them in children. The symptoms resembled those of ordinary primary nephritis. The patients lost appetite, were dull, and had a little evening fever for from 4 to 14 days. Then there was vomiting, dropsy, first of the face and then of other parts of the body, tenderness over the lumbar region, a diminution in the quantity of urine, which contained small quantities of albumen, and a few leucocytes and red blood cells. A moderate febrile movement continued through the disease. Cerebral symptoms were present in a few cases. The disease ran its course in about 14 days, but sometimes lasted as long as 6 weeks. The characteristic bacilli were found in great numbers in the urine. They were shorter and thicker than the tubercle bacilli, stained with methyl-violet, and could be easily cultivated. Twelve rabbits were inoculated with the cultures. Four of these developed a febrile movement, one died on the 14th day, the others were killed at the same time. In all of them there was ascites, and the kidneys were swollen and congested. In the kidneys, bacilli like those found in the urine, occurred in clusters in the stroma at the junction of the pyramidal and cortical portions of the kidneys.

Two autopsies were made in children. The kidneys were large and congested, numerous nests of bacilli were found in the cortex, and a few isolated bacilli in the tubes.

Forster[11] describes two curious cases of contracted kidney in two children, a boy and girl, the offspring of a syphilitic father and a mother with a diabetic family history. The boy, when 4½ years old, began to pass large quantities of urine of low specific gravity and without albumen or sugar. He became gradually more and more feeble and emaciated, and died when he was 9½ years old. The kidneys were found to be very small, with adherent capsules. The left ventricle of the heart was hypertrophied. The girl presented the same symptoms for three years and a half, and died when she was 8¼ years old.

Symptoms.—Three cases of profuse hæmorrhage from granular kidneys are related by Bowlby.[12] (1) A man, 73 years old, apparently bled to death from the kidneys. After death the prostate was found to be enlarged, the bladder was distended with pure blood, the kidneys were small and granular, there was blood in the pelves of the kidneys, in the ureters, in the renal tubules, and in the stroma. (2) A man, 49 years old, had profuse hæmaturia and died with uræmic symptoms. The kidneys were found small and granular. (3) A man, 64 years old, had profuse hæmaturia for several weeks, then this stopped and he had albuminuria and other symptoms of atrophied kidneys.

The phenomena which are commonly classed together under the name of uræmia continue to attract attention, although it must be confessed our knowledge concerning them is still very obscure.

Attention has been called during the past year to uræmic hemiplegia by Chauffard,[13] Raymond,[14] Lancereaux,[15] Suckling,[16] and others.

Attacks of uræmic hemiplegia are most common with the old and advanced forms of chronic Bright's disease, but not always with those which give marked renal symptoms; they may also occur with acute Bright's disease. They seem to be especially common in elderly persons with diseased arteries. The invasion of the hemiplegia is sudden, usually accompanied with coma. There is loss of motion alone, or of both motion and sensation. The hemiplegia and coma may continue up to the patient's death, or they may disappear after a few hours or days. In the latter case the patient may have several such attacks. These attacks have been ascribed to localized œdema of the brain, but certainly such an œdema does not always exist. They are undoubtedly more common in persons who have their cerebral arteries damaged by chronic endarteritis.

Coma, convulsions and mild delirium are well recognized features of uræmic attacks. It is not perhaps as well known that the delirium may be active and maniacal. This is especially seen when an old nephritis is complicated by an acute inflammation of some other part of the body. Headache, temporary loss of consciousness, temporary blindness, sleeplessness, vomiting, dyspnœa are well recognized symptoms.

The most hopeless cases of uræmia are those in which there is no sudden attack, no convulsions. The patient loses flesh and strength, he passes more and more time in bed, the mind becomes constantly more feeble, then come alternating stupor and mild delirium, and after weeks the patient dies.

Treatment.—The use of fuchsin to diminish the quantity of albumen in the urine does not seem to have met with much favor.[17]

The employment of calomel as a diuretic in bad cases of dropsy has been recommended by Jendrassik. The drug is given in doses of 0.2 gr. three or four times a day for three or four days, keeping the mouth washed with a solution of chlorate of potash. After two or three days there will be a sudden increase in the quantity of urine and then the calomel is stopped. When the urine is again diminished, the use of the calomel is repeated in the same way. If the calomel acts on the bowels a little opium may be given.

PHYSIOLOGICAL ALBUMINURIA.

The number of forms of albumen met with in the urine is considerable. They are described at some length by Dr. Stewart.[18] He distinguishes:—

I. *Serum Albumen*, a substance which, according to Hammarsten, constitutes 4.516 per cent. of the blood serum. It is almost constantly present in urine which contains any variety of albumen. Although a less diffusible body than serum globulin, it is capable of passing through membrane.

II. *Serum Globulin or Paraglobulin*, the globulin of the blood serum, of which it constitutes 3.103 per cent. It is met with in almost all albuminous urines, its proportion to the serum albumen varying in different instances.

III. *Peptone*, a product of gastric and pancreatic digestion of albuminous substances, also occurring in the process of transformation of tissues and of inflammatory effusions. It is a readily diffusible substance, occasionally met with in the urine in association with or apart from serum albumen.

IV. *Propeptone, Parapeptone, or Hemialbumose*, a substance or group of substances intermediate between albumen and peptone constituting a stage or stages of transformation from the one to the other. It is highly diffusible, and is occasionally met with in the urine under conditions corresponding to those under which peptone occurs. This is the peculiar form of albumen which was discovered in the urine by Dr. Bence Jones in a case of osteo-malacia.

V. *Acid Albumen or Syntonin*, one of the derived proteids obtained by the action of acids upon albumen. It is easily produced artificially by the addition of acid to albuminous urine, but may occur naturally in certain cases.

VI. *Alkali Albumen*, another derived proteid, produced by the action of alkalies upon albumen. It is readily produced artificially, but is also found naturally in the urine.

VII. *Hæmoglobin*, the combination of hæmatin and globulin naturally existing in the red corpuscles of the blood. It sometimes appears in the urine, particularly in cases of hæmaturia and hæmoglobinuria, also in certain septic conditions, and after inhalation of arseniuretted hydrogen, transfusion of blood, and otherwise.

VIII. *Fibrin*, a proteid substance which does not normally exist as such in the blood. It is met with in the urine in hæmaturia, in some cases of chyluria, and in certain varieties of renal casts.

IX. *Mucin*, the chief constituent of mucus, is a derived proteid substance. It frequently becomes superadded to the urine after secretion, and may be derived from any part of the urinary tract.

X. *Lardacein, Waxy or Amyloid Material*, familiarly known as a pathological substance within the body, is said to be occasionally demonstrable in renal casts.

Of these ten varieties the last four are evidently of little practical importance, mucin alone being indeed worthy of special comment, and that mainly because of the difficulties which its presence raises in regard to the reliability of certain tests for serum albumen.

As to the composition of the various albuminous substances, the statement of Hoppe-Seyler, that their percentage composition varies from C 51.5 H 6.9 N 15.2 S 0.3 O 20.9 to C 54.5 H 7.3 N 17.0 S 2.0 O 23.5, may be quoted.

TABLE I.—SHOWING TESTS FOR THE CHIEF FORMS OF ALBUMEN.

	Serum Albumen.	Serum Globulin.	Peptones.	Propeptones.	Acid Albumen.	Alkali Albumen.
Heat. Heat with HNO_3. Heat with A.	Opacity.	Opacity.	0	0	0	0 Opacity.
Cold, HNO_3.	Opacity.	Opacity.	0	Opacity dissolved by heat.	Opacity.	Opacity.
Metaphosphoric acid.	Opacity.	Opacity.	Opacity diminished or dissolved by heat.	Opacity diminished or dissolved by heat.	0	Opacity.
Acidulated brine . .	Opacity.	Opacity.	Opacity diminished or dissolved by heat.	Opacity diminished or dissolved by heat.	Opacity.	Opacity.
Picric acid	Opacity.	Opacity.	Opacity dissolved by heat.	Opacity dissolved by heat.	Opacity.	Opacity.
Pottassio-mercuric-iodide	Opacity.	Opacity.	Opacity dissolved by heat.	Opacity dissolved by heat.	Opacity.	Opacity.
Potassium ferrocyanide	Opacity.	Opacity.	0	Opacity dissolved by heat.	Opacity.	Opacity.
Dilution with water.	0	Slight opacity.	0	0	0	0
Magnesium sulphate.	0	Opacity.	0	0	Opacity.	Opacity.
Fehling's solution .	Brownish-red or mauve.	. . . .	Rose pink or purple.	Rose pink or purple.		
Randolph's test . .	. . . .	. . . .	Yellow opacity.	Yellow opacity.		

While it can hardly be said that there is any unanimity of opinion as to the causes of the production of albumen in the urine, yet the opinion of Semmola,[19] that it may be due either to changes in the kidneys, or to changes in the composition of the blood, or to changes in the circulation of blood through the kidneys, seems a probable one. And this would lead us to the conclusion that in physiological albuminuria the cause is to be looked for either in the composition of the blood, or in the character of the circulation of the blood; and this would accord with the conditions under which such albuminurias are developed, for they are found to be associated with changes of diet, digestion, position, and exercise.

The classification of physiological albuminurias into four varieties as proposed by Stewart[20] is a convenient one:

(1) *Paroxysmal or Cyclic Albuminuria.*—This is most frequent in young persons, especially males. The patients are often somewhat anæmic, poorly nourished, suffer from headache, neuralgias, bodily and mental languor, hysterical phenomena, and disturbances of the functions of the stomach, liver, and intestines. The albumen is regularly absent during the night and present at certain fixed hours during the day, although the time of day is not the same in every patient. Klemperer[21] describes the case of a young man in whom the albumen always reached its maximum quantity at 7½ in the morning and at 11 in the evening.

Teissier[22] describes 10 cases in which the albumen was only present at a time three or four hours after the first meal in the morning. As a rule no casts are found in the urine. In such cases the albumen may be increased by the erect position, by blowing on wind instruments (Sterling),[23] by mental and bodily exertion, by the ingestion of food. It may be made to disappear by keeping the patient in the recumbent position during the entire 24 hours. The most efficient mode of treatment is the improvement of the general health of the patient and relief of the disturbances of digestion.

(2) *Dietetic Albuminuria.*—This may occur at all ages. In some persons it only follows the ingestion of certain articles of food, especially cheese, pastry, and eggs. In others it follows taking food of any kind into the stomach. In others it will only appear when there are evidences of indigestion. In still others both food and exercise are required to develop it.

(3) *Albuminuria after Muscular Exertion.*—In these patients the albumen is absent from the urine except after muscular exertion. This exertion must be severe: boxing, athletic matches, long marches, etc. With this form of albuminuria casts may also be found in the urine.

(4) *Simple Persistent Albuminuria.*—This is a condition which may exist for years without other symptoms. The albumen is nearly always present in the urine. One may follow such patients for years and no other symptom of renal disease will make its appearance, and yet one never feels quite safe about such patients.

In all the varieties of physiological albuminuria the quantity of albumen is comparatively small. It is evident that, in their zeal to develop this comparatively new idea of an albuminuria without kidney disease, some authors are already confounding with it examples of true acute nephritis. The subject is of so much interest that the writer appends not only the references for the past year, but a number of others.[24]

INCONTINENCE OF URINE.

Several papers have appeared during the past year on the nocturnal incontinence of urine in children. Townsend[25] gives a table which represents very fairly our knowledge of the causes of this condition at the present time.

Causes of Incontinence of Urine.

I. Reflex.
  (1) Increased quantity of urine:
    (*a*) diabetes, (*b*) nephritis.
  (2) Irritant quality of urine:
    (*a*) increased acidity, (*b*) uric acid crystals, (*c*) calcic oxalate crystals, (*d*) excess of phosphates.
  (3) Vesical calculus.
  (4) Hypersensitive state of external genitals from:
    (*a*) stricture of urethra, (*b*) phimosis, (*c*) balanitis or vulvitis.
  (5) Anal irritation from:
    (*a*) pin-worms, (*c*) fissure, (*b*) eczema.
  (6) Psychical.
  (7) Increased irritability of bladder.
II. Atony of sphincter vesicæ.
  (1) General debility.
  (2) Spinal disease.
  (3) Acute febrile disease.
III. Malformations of bladder or urethra.

The treatment of this condition should be determined by its causation. If there is an adherent and inflamed prepuce, either the adhesions should be broken up, or circumcision should be performed. If there is increased irritability of the bladder, the use of belladonna and of the bromides is indicated. If there is atony of the sphincter, strychnia, ergot, and electricity are to be employed. If any one believes that, in these cases, there is congestion of the medulla oblongata, he may apply counter-irritation by cups or blisters over the nape of the neck.

TUMORS OF THE KIDNEY.

Adenoma, reported by Cornil.[26]—The kidney was removed during life. In the new growth were seen hæmorrhagic areas and calcified fibrous tracts; and budding into the cysts were gray, semi-transparent vegetations containing small, ovoidal cavities, lined by a single layer of epithelial cells.

Prudden has reported a case of multiple adenomata of the kidneys in a middle-aged man, who died from injury to the leg. Both kidneys were small, with adherent capsules, rough surfaces and thin cortices. The cortex of the right kidney contained a white, dense mass, wedge-shaped, and 1 cm. in diameter. Five globular bodies, some white, others reddish brown, softer than the contiguous kidney tissue, projected from the surface of the left kidney. They ranged from 7 mm. to 1 cm. in diameter, the capsule extending over them. They were composed of vascular connective tissue stroma, enclosing irregular tubular spaces, and filled with cuboidal cells. In some of these spaces were polypoid projections, covered with epithelium.

A large single adenoma is reported by Bouisson.[27] A male, aged 79 years, suffered from repeated attacks of hæmaturia for seven years, later with attacks of pain in the left lumbar region, and died in a condition of extreme exhaustion and emaciation. After death the left kidney was found to be converted into a nodular mass, 10 centimetres long and 6½ centimetres wide, which retained the general shape of the kidney, was soft, hæmorrhagic, and composed of spaces lined with epithelium, and polypoid growths covered with epithelium.

A myosarcoma striocellulare of the pelvis of the kidney and the ureter is reported by Ribbert.[28] The kidney was removed by operation from a female 4 years old. The pelvis of the kidney was enormously dilated. From the inner surface of this dilated pelvis and from that of the corresponding ureter there were given off numerous polypoid tumors. The tumors were composed of sarcomatous tissue, with numerous striated muscular fibres.

An atheroma of the kidney is described by Madelung.[29] The kidney was taken from the body of a man 22 years old. Only the lower third of the right kidney remained. Contained within the kidney, in a very thick capsule, was a large atheroma with calcified walls, containing small cysts with watery contents. These seemed to be retention cysts. The ureter was pervious, the suprarenal capsule was found to be somewhat atrophied, the left kidney was hypertrophied.

MOVABLE KIDNEYS.

Litten[30] distinguishes three varieties of displaced kidneys: (1) Simple displacement; (2) displacement with limited mobility; (3) the movable kidney. The second form is especially common in women, and is caused by lifting heavy weights, by tumors, by pregnancy, and by the pressure of the corsets. He calls attention to the frequency with which dilatation of the stomach produces the movable kidney on the right side. He found dislocation of the kidney in 17 out of 33 cases of dilatation of the stomach. The patients were mostly laborers who eat very quickly a midday meal composed of indigestible food.

RENAL CALCULI.

For the cure of renal calculi both medical and surgical procedures are employed. Ralfe,[31] in order to check the growth of calculi in the kidneys, advises the daily use of large quantities of distilled water, of benzoate of lithia and of turpentine in order to keep the urine of low specific gravity and to diminish the pyelitis.

Park[32] reports a successful case of treatment by much the same method in a man 30 years old, who had suffered from attacks of renal colic from the time that he was 11 years of age. During one of these attacks he was kept under the influence of morphia and atropia and made to drink very large quantities of fluid. He passed an oxalate of lime calculus weighing 14 grains and recovered altogether.

Bruen[33] reports a fatal case of renal calculus of long standing. The patient eight years before his death began to have pain in the left lumbar region with frequent and painful micturition. These symptoms occurred in attacks at irregular intervals. Eight months before death the attacks became more frequent and more severe and were attended with fever. The patient lost flesh and strength and died. After death there was found a large oxalate of lime calculus in the pelvis of one kidney; the pelvis of this kidney and the kidney itself were changed into a sac containing 13 oz. of pus.

Morris[34] reports a successful nephrolithotomy in a man 42 years old, who had given symptoms of a renal calculus for ten years. The calculus weighed 23½ grains, and was removed by an incision in the loin from the right kidney. The patient was discharged cured at the end of three weeks.

Marsh[35] reports a curious case of a woman who had an atrophied and movable kidney with two calculi in its pelvis, which caused much pain and discomfort. An incision through the lumbar region failed to discover the kidney. A second operation was performed by cutting through the anterior abdominal wall: the kidney was removed and the patient recovered.

Wright[36] reports two successful cases of nephrolithotomy. The first patient was a male, 28 years old. Five years before the operation he had an attack of pain in the right loin; for two years before the operation he had frequent attacks of pain, hæmaturia, vomiting and purging. The urine was of 1012 specific gravity, at first only contained mucus, later pus and .casts. The calculus removed from the kidney weighed 49 grains and was composed of uric acid. The second patient was a male, 30 years old, who for 21 years had suffered from attacks of pain in the right kidney. The urine contained blood, pus, casts and albumen. The calculus removed from the right kidney weighed 105 grains.

CYSTITIS.

During the year 1887 we find a number of papers in the French journals by Guyon,[37] Hartmann,[38] and others concerning painful cystitis. Under this name are grouped together all the examples of cystitis in which pain and tenderness are marked features, no matter what the other characters may be. The bladder is normally sensitive to contact and to distension. To contact its sensibility is very slight. The passage of a bougie is painfully felt up to the moment that the instrument enters the bladder; after this the contact of the bougie with the mucous membrane of the bladder is hardly felt by the patient. The distension of the bladder, on the contrary, as soon as it reaches a certain point, always gives rise to sensations and provokes the regular reflex contraction of the bladder. In certain cases of cystitis there is developed an acute sensitiveness to contact of the inner surface of the entire bladder, so that to touch it with any foreign body causes pain. In addition, the sensibility for distension of the bladder is exaggerated so that the presence even of small quantities of urine gives pain and excites reflex contractions. But to include a cystitis in this class of "painful cystitis" in addition to much exaggerated sensibility, there must be a persistence of it, a resistance to ordinary methods of treatment, a permanent contracture of the vesical muscle. Such a painful form of cystitis is most frequent with tubercular and gonorrhœal inflammation; less common with calculi, new growths and enlarged prostates.

The only effective treatment of this condition is by making an opening into the bladder, to be left open for some time, either above the pubes or in the perineum; and to treat the cystitis by cauterization, scraping, iodoform or washing.

RUPTURE OF THE BLADDER.

A remarkable case of recovery from rupture of the bladder is reported by Morris.[39] A man, 36 years old, after an injury developed symptoms of rupture of the bladder and of peritonitis, but after a month recovered. Six years later the cicatrix formed by the healing of the first rupture gave way and the patient died. After death there was found general peritonitis. A band 1¼ inches long united the fundus of the bladder with the rectum. The end of this band nearest the bladder was hollowed out into a funnel shape, and through a rupture in it close to the bladder, a string of mucus was projecting. Slight pressure caused purulent urine to ooze from the bladder through the rent into the peritoneal cavity. The case shows that an intra-peritoneal rupture of the bladder is not necessarily fatal.

Bennett[40] reports a case of extra-peritoneal rupture of the bladder due to aspiration. Taking this case as a text, he urges more caution in the aspiration of the bladder, and lays down the following rules:—

(1) Aspiration of the bladder, for the relief of the retention of urine, can only be resorted with absolute safety for cases in which the bladder walls are presumably healthy. (2) The operation is inadmissible in cases of retention consequent on long standing stricture, the bladder walls being diseased and often rotten; the appropriate treatment in such cases, if the stricture be impassable, being drainage of the bladder through a perineal incision. (3) The aspirator can be used only with some risk in cases of distended bladder where there is reason to suppose that the urine is foul, unless the organ is washed out with an antiseptic solution before the withdrawal of the needle. (4) In any case of bladder distension where there is reason to suspect disease of the coats of the organ, if tapping be performed at all, a large trocar and canula should be used, and not a small aspirator needle.

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URINALYSIS, CHYLURIA AND HÆMOGLOBINURIA.

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QUANTITY AND SPECIFIC GRAVITY.

Polyuria, Anuria and Oliguria.—Interesting observations on this subject by Robert Ultzmann[1] go to show that, beginning with infancy at about 300 ccm. in 24 hours, the quantity increases about 100 ccm. in the 24 hours for each year until the fifteenth, when the normal quantity is about 1500 ccm. in the 24 hours. Polyuria is only to be regarded as significant when constant. It is caused by the following conditions: (1) increased blood pressure through large ingestion of fluid, as in *urina potus*, or the polyuria attending the absorption of exudations; (2) nervous influence, as polyuria after section of the splanchnic nerve, in hysteria and diseases of the brain and spinal cord; (3) renal disease, such as the polyuria which usually shows itself at the beginning of improvement in acute renal diseases, and, according to Senator, as a result of the toxic action of urea. In chronic renal diseases the blood pressure increases either from cardiac hypertrophy or from the destruction of many glomeruli and renal tubes; (4) diuretics; (5) experiment.

Anuria is caused either by diminished arterial flow to the kidneys or obstruction at some point to the outflow of urine. The first may occur through irritation of the splanchnic nerve, which, according to Cohnheim, may take place in pregnancy. Hysterical anuria is a phenomenon frequently resulting as a reflex of derangement of the sexual organs in women, often relieved by treatment. To this category belongs sympathetic anuria of the sound kidney occurring in connection with disease of another kidney. Obstruction of the outflow of urine can take place (1) from obstruction of uriniferous tubules, as in cholera and all affections of the kidney; (2) through obstruction by stone, or by kinking or twisting of the ureter. A man 43 years of age, who had renal calculus, became suddenly anuric and died 14 days later. Autopsy revealed a renal cyst on the one side as large as a goose egg, and an obliterated ureter, while the other kidney was also enlarged, as well as its pelvis, and contained three small stones, completely obstructing the ureter. (3) Through a tumor of the urinary bladder.

Influence of Venous Stasis on the Secretion of Urine.—Paneth,[2] in a series of experiments, finds that a diminution of the secretion of urine occurs when the blood pressure varies from 1.5 to 6.4 mm. of mercury in the vena cava. The arterial tension diminished moderately after diminution of the lumen of the vena cava, but soon became again normal; and when the occluding ligature was beneath the liver and the portal system free from obstruction, the tension was normal. The effect of venous stasis was independent of any consecutive variation in the arterial tension. Ludwig had formerly explained the diminished urine in venous stasis by the mechanical compression of the urinary tubules by distended veins. If this was correct, Paneth thinks no diuretics could increase the secretion diminished by venous stasis, the reverse of which appears from the following experiment: the injection of nitre caused profuse diuresis to succeed lessened secretion, although the arterial tension was kept at a low degree by chloral hydrate.

The Specific Gravity of the Urine in Relation to Disease.—Charles W. Purdy[3] arrives at the following general conclusions: 1. That all structural diseases of the kidney causes a decrease in the quantity of salts excreted in the urine. 2. That if measured by the normal quantity of urine, 50 ounces, the specific gravity of the urine is decreased by all structural diseases of the kidney, referring here to the real specific gravity of the urine, *i.e.*, the specific gravity of the whole 24 hours' urine. 3. That the decrease of both the solids and the specific gravity bears a direct relationship to the extent of each and every lesion of the kidney. He says, further, that it even gives more trustworthy information than does the presence or quantity of albumin, as to the existence or otherwise of renal lesion; also as to its extent, its progress, its probable chronicity, and finally its progress toward recovery or death. 4. The specific gravity of the urine in functional albuminuria is never below the normal standard. The only exception to the above rules, of which Purdy is aware, is found in the concurrence of diabetes mellitus with chronic Bright's disease, wherein presence of sugar may so raise the specific gravity as to more than balance the lowering of specific gravity due to renal disease.

A Simple Method of Determining the Acidity of the Urine.—J. Michaux[4] suggests the use of officinal liquor potassæ, adding the alkali drop by drop to an ounce of urine, and measuring the degree of acidity by the number of drops required to neutralize the specimen.

ALBUMINOUS BODIES.

Tests for Albumin.—Henry B. Millard,[5] in a paper read before the New York Academy of Medicine, considers Roberts' nitric-magnesian test (1 part pure acid with 5 parts saturated sulphate of magnesium and filtered), as regards delicacy, accuracy and facility of employment, the most satisfactory test he has used. He however regards as equally sensitive his own test of phenic and acetic acid, and Tanret's mercuric iodide. He found the nitric-magnesian test to show less than one part in 1,000,000 of water; that is, .163 gram to the litre. It has the advantage of condensing the albumin more easily than does nitric acid. It cannot be used with urine clarified with liquor potassæ as a dense precipitate is at once formed, owing to chemical decomposition. In the discussion succeeding Millard's paper, Mendelson claimed that the method of boiling first, and then adding acid, was relatively the best test for albumin, emphasizing the boiling first. George B. Fowler and W. B. Thompson agreed with Mendelson. Charles A. Doremus regarded the ferrocyanide of potassium as sufficiently accurate while corroborative tests should be used in doubtful cases. The editor is satisfied that, all things considered, he can get the best and most reliable results by boiling and acidifying, great care being taken to work with a clear urine, made so by decantation, filtration, or such other method as circumstances suggest.

Serum Albumin in Normal Urine.—Posner,[6] by careful investigations, claims to have shown that a minimum amount of serum albumin is a constant constituent of normal urine. This may account for the infinitesimal quantities of albumin detected by such delicate reagents as potassio-mercuric iodide and picric acid. When we consider the constant presence in normal urine of albuminoid anatomical elements, it is not surprising that the disintegration and solution should liberate an amount of albumin which, though small, is still appreciable.

Esbach's Albuminometer.—This instrument consists of a glass tube graduated as figured in the text. The tube is filled to the mark U with urine, and then to the mark R with a test solution, consisting of picric acid 10 grams, citric acid 20 grams, water 1 litre. The tube is then closed with a rubber stopper and the contents cautiously mixed. The mixture is then allowed to stand undisturbed for 24 hours, and quantity of precipitated albumin read off. The reading indicates in grams the quantity of albumin in the litre.

Clinical Significance of Peptone.—Using the colorimetric method, Maixner[7] has made some observations upon the quantity of peptone in disease, with the following results: The greatest percentage was found in cases of empyema, .66 per cent.; in one case of pneumonia, .93; in another, .76 per cent. These high figures were only temporary, and showed that, even in cases most favorable both for the absorption of peptone from the blood and for its excretion in the urine, its amount reached but a fraction of 1 per cent. of the urine. The whole quantity in one day never reached five grams, whence the absolute loss of albumin is trifling; but the fact that it is lost in the form in which it is most easily absorbed, is one of some importance. It means that the organism has lost such a quantity of white corpuscles in the form of peptones that it is not able to reabsorb the whole amount. Maixner confirms his original view that the excretion of peptone runs a different course in different diseases. Thus, in inflammation of the lungs, peptone began to be excreted before the crisis. This is explained by the crisis having partially occurred, not in the main body of the lung affected, but in the parts first attacked and most advanced. The excretion of peptone hardly lasts two weeks, and in the third week, at most, it entirely ceases. The persistence and intensity of the reaction, and the quantity of peptone excreted, depends upon the amount of infiltration, the speed with which it dissolves, the age and station of the patient, and his state of nourishment. As a rule, during the crisis, the peptone was a little over three grams; after the crisis, one and a half to two and six-tenths grams per day. In a single case of gangrene of the lungs there was through the whole course of the disease the same amount of peptone,—from 1.99 to 3.66 grams per day. The increase of the excretion was easily explained by the spread of the disease and the secondary infiltration.

In pleural exudations, the amount of the peptone depends upon the amount of white corpuscles destroyed in the exudative fluids. The less the intra-thoracic pressure, the greater was the reabsorption of peptone. In one of the cases, the greatest amount of peptone was the day after tapping, being 4.96 grams,—the highest figure of the whole list. In suppurative peritonitis, owing to the large surface for absorption, the amount of peptone excreted was considerable. In cancer of the stomach, Maixner had no new cases to add to those already published. The amount of peptone discharged here depends upon various factors, as the spread of the tumor, the amount and site of the disease.

The Influence of the Peptonuria of Intercurrent Maladies upon Saccharine Diabetes.—Thorion[8] recalls the well-known fact that peptones prevent or retard the reduction of copper solutions by glucose, and suggests the following method of verifying it: The mixture of some drops of solution of peptone to the urine of the diabetic prevents the usual reaction. If to the urine of the diabetic one adds some urine from a case of pneumonia, the usual reaction is delayed for twenty-four hours, after which the red oxide is deposited; whence it is possible that glycosuria may be overlooked, or considered as less than it actually is if the urine contains peptones.

Thus may be explained the opinion commonly held that the presence of certain simple maladies, as scarlatina, pneumonia and acute intestinal affections suppress for a time the glycosuria. It is noted that the same result is obtained by the saccharimeter; in fact, peptones rotate light to the left, and the effect of the two reactions may exactly counterbalance each other. In the examination of 62 cases of this class, regarded as suppressing or diminishing the excretion of sugar, peptones were found 4 to 6 times, and 16 times the result was negative. In 13 cases of illness considered without reference to diabetes, intermittent fever, variola and pleurisy, the result was completely negative. Thorion concludes that in those cases the disappearance of glucose is solely apparent and fictitious. In order to determine it, it is necessary to leave the tube to itself for 24 hours. The negative reaction is possible, if the glucose is double the quantity of peptones, which is very frequently the case. The peptones may be eliminated by the previous action of hydrochloric acid.

The Existence of Pepsin in Urine.—Vasilevski[9] has found that both in healthy and diseased states pepsin, in greater or less quantity, was constantly present,—the smallest quantity being found in badly nourished patients, the nature of the disease having apparently less influence upon the amount of pepsin in the urine than the state of nutrition of the body. The least quantity of pepsin found was in the urine of a patient with pulmonary phthisis, four days before death, and in that of another who had carcinoma of the pylorus. In three cases where albumen was found in the urine the amount of pepsin was apparently the same as might have been expected had there been no albuminuria. A high body temperature appeared to lessen the pepsin.

GLUCOSE AND ALLIED SUBSTANCES.

Fehling's Solution prepared with Mannite.—M. Schmiedeberg[10] suggests the following modified formula for Fehling's solution, claiming that mannite insures stability: Dissolve 34.632 grams of crystallized copper sulphate in 200 cm. of water; add 15 grams of pure mannite in 100 cm. of water and 400 cm. of solution of caustic soda, sp. gr. 1145, and lastly sufficient water to make one litre. Use as Fehling's solution prepared in the ordinary way. The editor can fully confirm Schmiedeberg's claim to the stability of this solution, having had it in use many months without spoiling, while Fehling's solution made in the ordinary way spoiled in a comparatively short time.

Fehling's Solution made with Glycerine.—J. W. Holland[11] suggests the following as efficient, easily prepared and permanent: Cupric sulphate 1 drachm, glycerine 1 ounce; to make the test add 5 drops of the solution to 1 drachm of liquor potassæ in a test tube. Boil a few minutes to test the purity of the liquid, which should remain clear. Then add a few drops of urine. If glucose be present in quantity, there will be thrown down at once a red precipitate, as in the ordinary Fehling test. To detect minute quantities of sugar, after making the above, add half a drachm of the urine, boil and set aside. If sugar be present in very minute quantities, the liquid as it cools will become olive green.

Substances reducing Cupric Acid.—Among these are to be included turpentine, which when ingested, is converted into turpenoglycuronic acid, chloroform, benzoic acid, salicylic acid, camphor, which is converted into camphoglycuronic acid, of which the glycuronic acid has a strong reducing power, copaiba, cubebs, chloral, which is converted into urochloralic acid. Morphia also forms a reducing substance, as does also hydrochinon. Phenol and benzol are also converted into oxyphenic acid or pyrocatechin, probably identical with Boedeker's alkapton, and tannic acid into phenic acid, all reducing substances. To these may also be added oxybutyric acid, which being a substance deviating polarized light to the left, neutralizes to a certain extent the property of right-sided deviation possessed by grape sugar. Another is a carbohydrate discovered by Leo,[12] having the same empirical formula as glucose, dissolving cupric acid in the presence of an alkali, and reducing it on boiling. It does not, however, undergo fermentation, and deviates polarized light to the left. Its reducing power is about half that of grape sugar, and it is necessary to boil some seconds before cupric suboxide is precipitated.

Frank Donaldson, Sr., met in the course of life insurance examinations, a man apparently in perfect health, who declared he had never had any sickness in his life, whose urine threw down with Fehling's solution a copious precipitate of suboxide of copper. The man afterward came under his professional care, and numerous examinations of the urine were made. Finally he began to doubt whether there really was glucose in the urine. He then sent some to the editor of this department, who found the same reaction with Fehling's solution, but also found that after filtering the urine with animal charcoal, there was no response to Fehling's solution. He declared the reducing substance was not glucose. This was confirmed by Theo. G. Wormley and John Marshall. The latter declared the substance to be an unrecognized acid, and proposes for it provisionally the name glycosuric acid. T. Barton Brune, who also made a study of the substance, said that it was not pyrotocatecuic acid, although closely resembling it.

Modified Standard Solution for Johnson's Picro-Saccharimeter.—Johnson's standard solution being made of ingredients of the strength of the British Pharmacopœia, Chas. F. Adams[13] has substituted the following of the strength of the U. S. P.: Liquor ferri chloridi, 1 fluid drachm; ammon. carb., 1 drachm; ac. acet., 5 fluid drachms; aq. destillat., add fluid ounces, 3½. He has also changed the strength of the solution used in making the mixture to be heated, in order that the more convenient quantity of 5 cm. of each may be taken instead of the quantities given by Johnson. Thus of urine 5 cm., liquor potass. (sp. gr. 1036) 5 cm., solution of picric acid (grains 3.5 to a fluid ounce) 5 cm., water 5 cm. These are boiled for 60 seconds and the comparison made in the saccharimeter figured in the text as directed by Johnson.

Reducing Substance of Picric Acid in Normal Urine.—The earliest announcements by George Johnson of the revival of the picric acid test contained a statement that when normal urine is treated with picric acid, potash and heat, a slight coloration takes place, which Johnson estimates as about equal to the change which would be produced by a solution of glucose containing 0.5 to 0.7 grains to the fluidounce, which he thought might be due to some substance allied to sugar. Almost immediately after this announcement was made, Theodore G. Wormley taught at the University of Pennsylvania that the reducing substance was creatinin. In 1886, Jaffe[14] announced that it was a mixture of uric acid and a double salt of picrate of potassium and picrate of creatinin. Within the past year Johnson[15] concludes, as a result of some recent researches of his son, (1) that not a trace of glucose is found in normal urine; (2) that as uric acid has no reducing effect on picric acrid, the chief, if not sole constituent, of normal urine which reduces picric acid in the presence of potash is creatinin. He has discovered a simple method, to be subsequently published, of precipitating creatinin from urine in combination with a metallic salt, and by a process which has no effect upon any glucose present. A specimen of normal urine when thus treated ceases to give a red color when boiled with picric acid and potash; but after the separation of the creatinin, if glucose be added to the urine in the proportion of a grain to the ounce, the sugar is found in diminished quantity after the creatinin has been removed. Johnson also announces that the urine of patients who are taking salicylate of sodium when boiled with picric acid and potash, gives a deeper red color than is quite normal, the color being that which would result from the presence of one to two grains of glucose per ounce. He says that Fehling's solution would also be reduced as it would be by a small amount of sugar. His son has been investigating this substance, and finds that it is neither glucose nor an excess of creatinin, but is probably due to some product of the metabolism of sodium salicylate, the precise nature of which is unknown. Salicylate of sodium itself has no reducing effect.

Fermentation Test.—Ottoman Rosenbach[16] regards fermentation as theoretically the most reliable means of recognizing minute quantities of sugar, and has adopted its principle for the practical purpose of detecting sugar in the urine. A small quantity of urine, the incomplete reducing effect of which, in a measured quantity of Fehling's solution—1 cm. of urine in an equal volume of Fehling s solution,—shows the presence of less than a half per cent. of sugar, is boiled after the addition of a few drops of tartaric acid, which prevents the precipitation of the phosphates. The urine after cooling is divided into two portions. To one of these a small quantity of yeast is added and this portion is kept in a warm place. If after two hours equal parts of each specimen be taken and tested with equal quantities of Fehling's solution, one of them will reduce the solution, while the other, proportionate to the degree of decomposition of the sugar, will give either a perfect negative, or at any rate a noticeably weaker result. By this method Rosenbach says that extremely minute quantities of sugar will be detected with certainty, as he claims to have shown by experiments on diabetic urine and normal urine when sugar was added. The presence of albumin does not interfere with the application of this test. When it is remembered that urine or any fluid holding .4 per cent. of sugar will absorb all the carbonic acid generated in the fermentation, it is difficult to comprehend how this, among other claims to such extreme delicacy in the fermentation test, can be substantiated. This fact seems to have been entirely overlooked by Max Einhorn[17] and partly by S. P. Kramer,[18] who describe and figure what they regard as the improved instruments for the recognition of minute quantities of sugar by the fermentation test,—the former claiming to be able to recognize $\frac{1}{10}$ of 1 per cent. of sugar, and, after boiling, $\frac{1}{20}$ of 1 per cent.

The Phenylhydrazin Test.—This test, suggested by Emil Fischer, is claimed to be exceedingly delicate. We have not the original directions, but very clear are those furnished by T. C. Van Nüys in his recent work on Chemical Analysis. Fifty ccm. of the suspected urine are put into a beaker, and 2 grams of phenylhydrazin hydrochlorate, with 1.5 grams sodium acetate (or 1 gram of the latter if the urine is not decidedly acid). Unless the urine is nearly colorless, 20 cm. of water are added. The beaker is then placed in a water bath and gently warmed for one hour. If sugar is present, needle-shaped crystals of phenylglucosazon will form. These must, however, be identified by their melting point, 204° to 205° C., as well as by the microscope. The former is accomplished by separating the crystals by filtration, washing with a small quantity of water and dissolving in a small amount of dilute alcohol, and evaporating at a low temperature, when the substance will again crystallize out. This process is repeated two or three times, when the crystals are dried in a desiccator over concentrated sulphuric acid. A piece of thin glass tubing is then drawn out in a flame, so that the sealed capillary extremity is 2 or 3 cm. from where the tube is of its original diameter. The tube is broken by a file near where the contraction begins and a small portion of the dry substance introduced into the sealed extremity. The piece of tubing is now attached to a thermometer by means of a rubber band. The capillary end of the tube containing the substance is placed adjacent to the bulb of the thermometer, the tube with the bulb is introduced into concentrated sulphuric acid, and the acid gradually heated. As the mercury ascends to 204° C., the substance will begin to show evidence of fusion, provided the increase in temperature be gradual and the heat be equally diffused by stirring the sulphuric acid with a glass rod. This test is strongly commended by von Jaksch. It is said not to react with other reducing substances which occur in the urine; but albumin, if present, must first be removed. The reducing urines passed after the administration of benzoic and salicylic acid, and in cases of poisoning by potassium hydrate and sulphuric acid, do not contain sugar. Traces of sugar were found in the urine in three cases of carbon dioxide poisoning, and in two cases of asphyxia following the inhalation of other poisonous gases.[19]

A. K. Bond[20] publishes a ready method of using this test, suggested by Ultzmann, of Vienna, together with some results of his observations, which go to show its extreme delicacy and positive character. Pour the phenyl salt, which is a dry substance resembling bran, into an empty test tube to the depth of about four-tenths of an inch, and add crystals of sodium acetate ground fine, to an equal height. Upon this pour the urine, whether clear or cloudy, until the tube is half filled. This gives in a test tube five inches long, about the following proportion in weight, viz.: 1 part phenyl salt, 2 parts sodium acetate, 15 parts urine. Shake the tube until the crystals of sodium acetate are dissolved; then heat gently over a low flame until the mixture boils, and boil it for half a minute: whether it becomes clear or not, makes no difference. Then cover the tube and let it stand, and after a proper interval examine the sediment with a microscope. If the sugar is present, there will be seen first fine bright yellow needles, each branched out or joined by others as they are formed, till the field is dotted with groups like delicate sprays or sheaves, all radiating from the centre. A magnifying power of 200 diameters is sufficient for this study. That phenyl salt is always in excess when the test is made in this way, is shown by the constant presence of reddish globules in the field. The following are Bond's conclusions: (1) The test of Fischer, as applied by Ultzmann, is very easy to make and does not take more time than Fehling's, if, as is necessary, he says, the latter be made with fresh solutions. (2) It is suitable for office use by physicians, and may be accurately made by those whose training in chemistry is imperfect. (3) It does not require great skill in the use of a microscope. In all of these examinations, the crystals, if present, are found upon the first slide examined. (4) When the sugar is present, to the amount of one-fifteenth per cent., the crystals may be found 15 minutes after the test is made. (5) The test is about equal in delicacy to Fehling's, is not liable to be disturbed by any constituent of the urine, and is obtained in alkaline as well as in acid, in cloudy, as well as in clear urine. (6) The normal urine does not respond to the test. (7) If crystals are found, the proof of the presence of sugar is absolute. (8) Bond's experiments lead him to believe that in all cases in which sugar is present to the amount of one-fortieth per cent. or more, the crystals may be found, although it may be necessary when the quantity of sugar is very small to let the mixture stand after boiling as many as 48 hours. (9) Single crystals show a tendency in the most minute as well as in the most concentrated urine, to form clusters, so that in solutions which have stood for some time, only the charactistic clusters may be found. (10) The crystals or clusters undergo no change of importance in aqueous solution for a month or more, and so any sediment may he preserved and inspected from time to time. Often in urine mixtures, they remain for weeks unchanged, decomposition of the mixture being prevented by the phenyl salt. The advantages claimed for this test, if confirmed, seem very great indeed, and its availability is much increased if Bond's method be sufficient; but he is hardly correct when he says that Fehling's solution should be made fresh for each testing, since the latter can be kept fresh a long time, with ordinary precautions, and with the modified formulæ for its preparation that have been suggested, and which greatly aid in its preservation. Indeed, if it had to be made up fresh each time, it would soon pass into disuse as an inconvenient test. In our judgment the testing by a good Fehling's solution is far more convenient.

Alphanaphthol and Thymol Tests.—As directed by Molisch,[21] ½ to 1 cm. of the fluid to be tested is mixed with 2 drops of a 15 to 20 per cent. alcoholic solution of alphanaphthol, and an excess of concentrated sulphuric acid added. Upon shaking, if sugar is present, a deep violet color is developed, and upon dilution with water, a violet blue precipitate occurs, soluble in alcohol and ether, with a yellow color, or in potassium hydrate, with a deep yellow color. If the alphanaphthol is replaced with thymol, a deep red color is produced, and on dilution with water a carmine-red flocculent precipitate, soluble with a pale yellow color in alcohol, ether, and potassium hydrate, or with a bright yellow color in ammonium hydrate. Most varieties of sugar respond to these reactions. Inosite, however, does not. Most glucosides respond sooner or later. Indican is an exception. Urea, creatinin, xanthin, uric acid, allantoin, hippuric acid, succinic acid and, finally, pyrocatechin, all give negative results. This test according to Molisch is far more delicate than any other known test for sugar, detecting the latter in solutions containing only .00001 per cent. Normal urine responds to this test even when diluted with 300 volumes of water. Molisch considers, therefore, that the presence of the sugar as a constant constituent of normal urine, can no longer be doubted. To distinguish normal from diabetic urine, he recommends the following proceeding:—(1) Dilute the specimen of normal urine and one of the urine to be tested for sugar with 100 volumes of water, and compare the colors resulting on the application of the test. (2) Dilute two similar specimens with 300 or 400 volumes of water. The diabetic urine will still respond to the test, while normal urine will fail to respond under this degree of dilution.

Seegen has submitted this test to careful examination with the following results, viz., sugar solutions containing. 05 per cent. gave both reactions distinctly. Solutions containing .01 per cent. of sugar gave with thymol a dark sherry yellow color, with alphanaphthol a faint violet tint, but on dilution with urine, no precipitate was formed. The tests are therefore less delicate, according to Seegen, than are Trommer's. Normal urine gave the reaction as described by Molisch. When urine was diluted 100 times, a light red or violet color was obtained, but no precipitate. Urea and uric acids gave negative results with both tests. But various familiar substances and secretions and chemically pure albuminous bodies, for example peptone, egg albumin, serum-albumin, and casein all gave the reactions. These reagents, according to Seegen, are of no value as tests for sugar alone, and Molisch's conclusion that sugar is a constituent of normal urine is not justified. In answer to Seegen, Molisch maintains that his reagents are more delicate as tests for sugar than Fehling's solution. In dilute solutions it is necessary to employ a small quantity of the solid alphanaphthol in place of the naphthol *solution*. With reference to albuminous bodies, Molisch says that while these may give results resembling somewhat those obtained by sugar solutions, still the precipitates obtained upon dilution with water are, except in the case of peptone, of different color—dirty yellow or yellowish brown—from those produced with sugar solutions. Besides, they are all soluble in hydrochloric acid with carmine red or reddish violet color, while the precipitate obtained by the sugar solution is insoluble in hydrochloric acid. Molisch now maintains that hydrochloric acid in place of sulphuric acid may be employed in these tests, the mixture being subsequently boiled for a minute. Fibrin, vitellin, serum albumin, egg albumin and peptone do not give the reaction when hydrochloric acid is used. Normal urine gives a reaction when boiled with alphanaphthol and hydrochloric acid, even if diluted 10 times. Molisch still asserts that normal urine must contain sugar, or else something as yet unknown.

Detection of Small Quantities of Glucose.—Mehu[22] recommends the following: Evaporate the urine to a syrupy consistence over a water bath; after cooling, treat with 90 per cent. alcohol. The liquid is evaporated again after filtration to drive off the alcohol. Then the residue, redissolved in water, may be tested. The creatinin, which is a great inconvenience, may be removed by adding to the alcoholic liquid a little chloride of zinc, but it is necessary to wait 24 hours before filtering and making examination. Mehu also advises in doubtful cases to add to the urine one-twentieth volume of a solution of basic acetate of lead such as is found in the shops, and filter. Add to the filtrate powdered anhydrous neutral sodium carbonate, and after agitation and separation of the plumbic precipitate, concentrate the decolorized liquid to one-tenth its volume and filter. If Fehling's solution does not respond, you may consider sugar is absent. Since these methods, although simple, are apt to occupy a great deal of time, Mehu recommends especially the method of Worm-Müller, which is that of Fehling modified. Two solutions are necessary, one of 2½ grams sulphate of copper in 100 grams of water; the other, 10 grams of the salt of Seignette, and 4 grams of soda in the same quantity of menstruum. Heat separately in two tubes to ebullition, 5 ccm. of urine, having first removed any albumin if present, and a mixture of 1½ to 3 ccm. of solution of copper, 2½ ccm. of the alkaline solution. The action of the heat is interrupted at the same time, and after having waited 20 to 25 seconds, the two are united. The fluid becomes bluish green and decolorizes rapidly, the oxide of copper remaining generally in suspension.

The density of the urine serves as a guide as to the quantity of sulphate of copper to be employed. When it exceeds 1020, use only 2 ccm.; when it is less than this, use 1½ or 1 ccm. The rate of the reaction varies with the proportion of the sugar. When this is less than .05 per hundred, it is necessary to wait 4 or 5 minutes, when the cuprous precipitate becomes visible, forming then a doubtful yellowish green. The clearness of the reaction may be obscured by a deposit of phosphates, which forms even at the moment of mixture. This deposit produces a yellowish green color, different from the well-known yellowish reddish color of the cuprous precipitate; but in case of doubt it is necessary only to wait some minutes. Almost always the phosphates deposit rapidly, while the cuprous oxide remains a long time in suspension.

Laache, who repeated these operations, is able to detect by the aid of this process .025 per hundred of grape sugar and .05 of sugar of milk. The proportion of a .1 per hundred is unmistakable. Mehu says it is the exception that a urine which does not contain sugar contains a substance which produces this reaction. Laache advises that if the reaction is doubtful, the patient may be made to take for some days starchy and saccharine food. If the reaction continues absent under these conditions, we may eliminate the idea of glycosuria.

Baas, for quantities of .1 per thousand and below, proposes to use a piece of filtering paper impregnated with indigo and another of carbonate of sodium, which are preserved dry in a portfolio. To the urine contained in a test tube or capsule, a portion of each paper is added. In a case of glycosuria the blue color which is produced disappears rapidly, and the liquid takes a clear yellow color, or remains completely colorless. This procedure recommends itself by its simplicity, but is not regarded by Mehu as reliable. This is evidently the indigo carmine method of Oliver, of London. According to Mehu, his method permits the elimination of almost all the following substances which are said to reduce salts of copper, viz., turpentine, which is said to excite the secretion of a substance which reacts with bismuth, with yeast, and Fehling's solution, and which is optically negative; lactose, which is said to be secreted sometimes in large quantities by nursing women; so also uric acid, glycuronic acid, creatinin, allantoin, lucin, indican, and the products which appear after the use of oxalic acid, benzoic acid or chloral.

A Starch-converting Ferment in Human Urine.—Holovtschiner[23] recently demonstrated in normal and pathological urine a ferment which possesses the same power as the ptyalin of saliva, of converting starch into dextrine and sugar. He added to 10 ccm. of urine 5 ccm. of a 1 per cent. solution of boiled starch, and placed the mixture for 4 hours in a water bath at 40 degrees C. After a time he found the starch no longer demonstrable, while the Moore-Heller test discovered sugar. The subject has been still further investigated by R. Breusing,[24] who repeated the experiment, placing the mixture in an oven. He confirmed Holovtschiner's results to a certain extent, but failed to obtain response to the fermentation test, while Trommer's test also gave negative results.

Lactosuria.—Leon Arturo[25] defines this condition as the elimination by the kidney of mammals of sugar of milk, which, not being used in lactation, is reabsorbed by the blood-vessels and lymphatics and reappears in the urine. Lactose is not absorbed before the seventh month of pregnancy. It is constant in the last two months, and corresponds with development of the milk glands. The woman will be a better wet-nurse the more at the beginning of pregnancy lactose is found in the urine, and the greater the quantity in the eighth and ninth month. In women who do not nurse, the lactosuria is constant in the five or six days which follow delivery and then disappears completely. With women who nurse we find lactosuria in the first four days, but the quantity of sugar is small, amounting to 1–3 grams per litre. If the woman suspends lactation from any cause, the lactosuria ceases after some days.

Acetonuria.—In a joint paper on this subject by Moscatelli and Vitali, the former publishes the results of examination of large quantities of urine of healthy individuals. In no case did he find a trace of acetone. Hence he regards it as always of pathological import. Vitali relates two cases, in which during life acetone appeared continuously in the urine and for a long time. One was that of a diabetic woman of 35, who had a distinct sour-milk odor from the breath. Acetone was found with both Lieben's and Legal's tests, regardless of diet. Toward the end of life the patient had an attack of erysipelas, during which both sugar and acetone disappeared from the urine. The second case was one of severe intestinal obstruction, arising from malignant tumor, in which regularly every day there was vomiting of copious fetid darkish brown masses, while the stools contained only a small quantity of hardened fæces. The breath had an ether-like smell, the urine was scanty, high colored, and contained large quantities of acetone, which was found also in the vomited matter. In neither of these cases was there a symptom of acetone intoxication. In a healthy individual, only when 8 ccm. of acetone had been administered could Vitali find any in the urine.[26]

Acetonuria and Diaceturia.—Rossbach[27] relates the case of a diabetic patient, 23 years old, living upon a pure albumen and fat diet with total exclusion of the carbohydrates. The weight increased from 89 to 107 pounds. The daily urine sank from 4000 to 2000 ccm. During all this time acetone and diacetic acid were demonstrable in the urine. After four days use of amylaceous food, the urine ran up to 3600 ccm., the sugar to 7 per cent., while the acetonuria and diaceturia disappeared completely. Rossbach hence concluded, contrary to Von Jaksch, that acetonuria and diaceturia may be simultaneously present in the same urine; that along with the apparent improvement in the patient, the diaceturia had no prognostic significance; and that, finally, an absolute albuminous diet could become a cause of acetonuria and diacetonuria.

In a recent paper, J. P. Crozer Griffith[28] clearly indicates the difference in the clinical significance of these two conditions. As a rule, the presence of acetone in the urine need cause no special anxiety. On the other hand, the presence of diacetone is always a grave symptom, except in some cases in children. It is therefore very important to know of its presence on account of its prognostic indications. In the present state of our knowledge, according to Griffith, we are unable to hinder the development of diacetic acid in the system, or to remove it when formed. Both acetone and diacetone may occur in the urine under the same circumstances, among which are febrile states, diabetes, carcinoma, inanition, psychoses and idiopathic conditions.

Baginsky[29] has come to the following conclusions regarding acetonuria in children: (1) Acetone occurs in the urine of healthy children under completely normal conditions, but in very small quantities; (2) in febrile states in children in a great variety of affections, as pneumonia, measles, etc.; (3) it increases with fever and diminishes with its decline; (4) it is probably produced from the disintegration of nitrogenous matter in the organism, for it is increased in the dog fed on highly nitrogenous food, and can be diminished in the same animal by continuous feeding with carbohydrates, even to complete disappearance; (5) acetonuria is not increased by dyspnœa, and artificial dyspnœa in animals being unattended with acetonuria. Even in severe asphyxia produced by carbonic oxides, acetone does not appear in the urine; (6) acetonuria is enormously increased in children with eclamptic attacks,—sudden epileptiform convulsions. The source of this acetonuria cannot be found in a coincident medication by chloral hydrate; (7) the source of this acetonuria is also not to be sought in the fermentation phenomena in the intestinal canal; at least in the lactic acid fermentation the quantities of acetone are very insignificant; (8) neither in the fæces, nor in the contents of the stomach, can acetone be shown to be present in dyspeptic children in a single exception being noted; (9) the presence of acetone cannot be regarded as the cause of eclamptic seizures in children, for in the diseases in which convulsions are common, acetone is either altogether absent, or is found in minute traces; (10) the suspicion that acetonuria stands in any relation to rickets is not confirmed by clinical or experimental facts. The dog long fed with acetone remains free from rickets. Neither is nephritis produced in animals by acetone, in opposition to the statement of Albertoni and Pisenti.

Diabetic Lipaceduria and Lipacidœmia.—R. von Jaksch[30] investigated diabetic urines with a view to the detection of volatile organic fatty acids. Of eight cases affected partly with acetonuria and partly with diaceturia, in one only was lipaceduria present, and this presented no symptoms unusual in this condition. In the blood of diabetics were occasionally found traces of volatile fatty acids, without demonstrable diminution in its alkalescence. Only when there were large quantities of fatty acids and acetic acid in the urine did the alkalescence of the blood appear diminished. This diminution in the alkalescence of the blood at the same time at which the volatile fatty acids and acetic acid are found in the urine, and moreover the presence of volatile acids in the blood and urine, constitute, according to von Jaksch, a further confirmation of the view of Naunyn and his school,—that acid intoxication is a not unimportant factor in the diabetic process. Since, according to Meyer, the secondary fatty acids produce a toxic acid in the organism, it is possible that a part of the symptoms observed in diabetic coma are produced with the formation and absorption of a large amount of volatile acids. Further investigation of the fatty acids in diabetic coma are, however, necessary to enable us to come to conclusions on this point.

COLORING MATTERS.

Urobilin and Urobilinuria.—Hayem[31] has reported to the Société des Hôpitaux, of Paris, some important observations on urobilin, the hemaphéine of Gubler, which is recognized in the blood and urine by the spectroscope. The spectrum is characterized by an absorption band between the lines B and F. The urine always contains a small quantity of urobilin, and urobilinuria is only an exaggeration of the physiological phenomena, acquiring importance only when constant. Urobilin communicates a deep red hue to urine, varying with the quantity present. It exists alone, or associated with other biliary pigments. It is always met with in the period of asystole in cardiac diseases, frequently in diseases of the liver, including cirrhosis, cancer and steotosis, and in the intoxications, especially of lead and alcohol. Among other affections may be mentioned rheumatism, acute gout, pneumonia, gastric embarrassment, angina and intermittent fever. Whenever Hayem has observed albuminuria, he has found a venous stasis of the liver, with a slight catarrh of the biliary ducts and infiltration or fatty degeneration of the liver cells. As to the origin of urobilin, Hayem believes that a turgid liver, instead of producing normal pigments, manufactures urobilin, which passes from the biliary organ into the blood. He considers it the pigment of disease of the liver, and a sign of hepatic insufficiency. Permanent urobilinuria has an immense importance in prognosis and diagnosis, as it indicates infiltration or fatty degeneration of the hepatic cells.

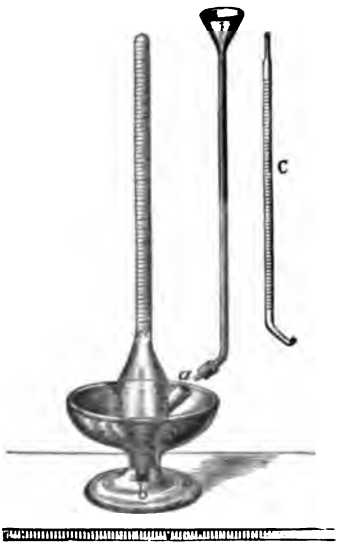
Keener and Engel[32] have also made observations on the same subject, and conclude that the urobilin which appears in the urine under certain pathological conditions proceeds from biliary coloring matter deposited in the tissues; that the formation of urobilin may be considered as the most advantageous means by which the organism can get rid of the soluble and diffusible biliary pigment, which has a great tendency to fix itself in the tissues in the form of drops and granules, whose accumulation interferes with the vitality of the anatomical elements; that urobilin is, on the contrary, diffusible in the same way as crystalline substance like urea, and ought by this property be eliminated by the urine.

The Hæmapheic or Urobilin Reaction.—It is well known that the contact of nitric acid with certain kinds of urine in icteric patients often produces a deep brown color, shading gradually off as it is farther removed from contact with the nitric acid. The play of bile colors is only observed in certain patients. Extensive experiments by Engel and Keener go to show that it is not due to one substance, but to the blending of coloring and chromogenic matters contained therein. The hæmapheic reaction is produced by the contact of nitric acid with the urine, if this contains sufficient chromogens to deepen the color which it naturally has.[33]

New Coloring Matter in Urine.—Leube[34] reports finding a coloring matter in the urine of those suffering from osteomalacia, cystitis and nephritis, which turns on exposure to the air from dark brown to black. This amorphous substance dissolves in ether. It is separated by dilute alkalies, not by acid. The alkaline solutions are first reddish brown, afterward yellow. It is soluble in hot water, chloroform and benzole. The solution is decolorized by zinc, but its color on exposure is restored. The solutions show the characteristic spectra or efflorescence.

UREA AND ITS ESTIMATION.

Apparatus for the Estimation of Urea by the Hypobromite Process.—John Marshall[35] has devised an apparatus in which the measuring tube is easily filled, removed and cleaned. The inferior oval extremity, with its attached measuring tube, holds about 77 ccm., and can be fastened in a saucer-like glass vessel by means of a perforated cork as shown in the figure, so that the overflow of the hypobromite solution during the operation may be collected. The apparatus can be taken apart easily and cleansed of the grease-like material which accumulates in the tube during the process.



MARSHALL'S APPARATUS.—(*Zeit. f. Med. Chemie.*)

In using the apparatus the thumb is placed over the opening *a*, and the hypobromite solution is poured in through *b*. The latter opening *b* is then closed with a rubber stopper, and any air bubbles in the tube are allowed to pass out at the opening *a*. The graduated tube is now reversed, and the oval end of the tube is fastened in the opening in the cup-like vessel shown in the figure. The urine under investigation is, by means of the pipette c, passed into the hypobromite solution in the tube through the opening *a*. One cubic centimetre of urine generally suffices for the operation. Decomposition of the urea immediately occurs, and the evolved nitrogen collects in the upper part of the graduated tube.

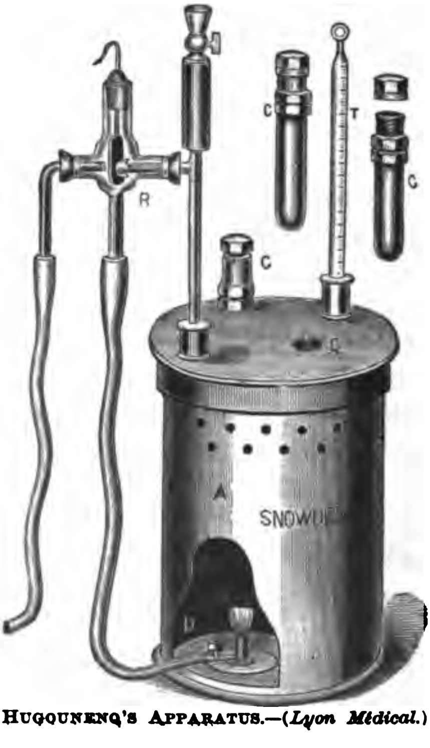
About 20 minutes are required for the decomposition of the urea, when, all the gas bubbles having collected with the gas in the upper part of the tube, the atmospheric pressure is equalized by attaching the funnel tube to the opening *a* and pouring into it hypobromite solution or water until the surfaces of the liquid in both tubes stand at same level. The number of cubic centimetres of nitrogen, the temperature and barometric pressure are read off, and the percentage of urea calculated by the following formula:—

$$p = \frac{100.\ v.\ (b-b')}{760.\ 354.33.\ a.\ (1+0.00366\ t.}$$

In which

p represents the weight of urea in grams in 100 c.c. of the urine.
a " " volume of urine employed.
b " " observed barometric pressure in millimetres.
t " " observed temperature (centigrade).
v " " volume of nitrogen, in cubic centimetres, obtained.
b′ " " tension of aqueous vapor for the temperature t.

A New Apparatus for the Determination of Urea in the Fluids of the Organism.—Like all organic bodies which belong to the class of amides, urea heated in a closed vessel at a temperature sufficiently elevated and in the presence of a great excess of water, hydrates itself and furnishes a corresponding ammoniacal salt, carbonate of ammonium. For the estimation of this by the alkalimetric method, Cazeneuve and Hugounenq[36] have devised an apparatus figured in the cut. In the interior of the cylinder of copper is placed an oil bath, also of copper. The bath is heated by the flame D, and brought to the temperature measured by the thermometer T, maintained constant by the regulator R.



Hugounenq's Apparatus.—(*Lyon Médical.*)

In the oil of the bath extend two large tubes of bronze lined with platinum, closed at one end and tested to bear a pressure of above 60 atmospheres. The upper part is made to permit a metallic cover to be firmly screwed, a washer of lead protecting the bottom of the cover to assist in securing a hermetical seal. Two keys joined to the apparatus govern the screw, and permit the exact opening and closing of the tubes. To use the apparatus, we first take about 25 to 30 ccm. of urine. This is agitated for a time with bone-black and filtered. The urine passes through decidedly decolorized, though less effect is produced on strong pigmented urines; but the alkalimetric process is not thereby interfered with. Of the urine thus treated, exactly 10 ccm. are run into tube C, about 20 c.c. of distilled water are added, and the cover is screwed on with care. Heat is applied to 180° C. At the end of a half hour of heating, the tube is permitted to cool. The liquid with the wash water from the tube, is collected, and the alkalimetric test is made. The alkalinity of the liquid is determined with a normal solution of sulphuric acid, one litre of the normal solution containing 40 grams of SO_3. Aniline orange No. 3, or phenol phtalin is employed as the indicator. It suffices to multiply by 3 the number of cubic centimetres of the acid solution employed to have expressed in grams the quantity of urea in a litre of urine. The result of comparative trials of this method with Liebig's method, and the hypobromite process on solutions of pure urea, is exhibited in a table, which appears decidedly in favor of the alkalimetric process. The difficulties in the case of mixed solutions like urine are explained, but no results are given.

ACIDS OF THE URINE.

Clinical Significance of Uric Acid Deposits.—In some recent observations by Johannes Mygge,[37] uric acid sediments were found in large quantity in 262 specimens derived from 59 patients. In 43 of these last patients the deposits were of a transitory character,—that is, they were only observed once or twice, while in the remaining 16 they persisted a week or more. Deposits both of a transient and permanent character were found especially in rheumatic affections, whether acute or chronic. Transient deposits were also found in pneumonia in 11 cases out of 25. In 27 out of 59 patients in whose urine uric acid deposits were found, albuminuria was also found in appreciable quantity, and in many of the rest doubtful traces were noticed. In quite a number of cases the lithæmia was seen coincident with albuminuria. Frequently, too, uric acid appeared as the acute albuminuria ceased, and in many other lithæmic cases, microscopic examination revealed the presence of tube casts and renal epithelium. Mygge is convinced that there is frequent, if not constant, relation between the renal affection and persistent deposits of uric acid, though unable to explain the nature of this relation. Albumin may be produced in some cases by a prolonged action of the highly acid urine on the tubules. On the other hand, the precipitation of uric acid may be produced by morphological elements existing in the urine coming from kidneys already diseased. Uric acid sediment also appears to bear some relation to the desquamation of vesical epithelium and certain forms of incontinence of urine.

The association of albuminuria and uric acid sediments, with or without hyaline casts, has frequently been observed by the editor in urine of high specific gravity and acid reaction, and he has come to regard the albumin and casts as the result of the irritating effect of the acid urine on the tubules.

New Acids found in Urine—Glycuronic Acid.—Schmiedeberg and Meyer[38] found in the urine of dogs, to whom camphor had been given, an acid named by them glycuronic acid, on account of its relation to the carbohydrates. It is a coupled acid, reducing alkaline solutions of copper, deviates polarized light to the left, and has the formula $C_6H_{10}O_7$. The same coupling is found in the urochloral acid obtained by Mikulicz and von Mering out of chloral urine, as well as in the acid obtained by Jaffe from the urine of dogs, after the administration of orthonitrotoluol. Glycuronic acid itself is a non-crystalline syrup. It possesses a sweetish, somewhat bitter taste. It turns polarized light a D+19.25. Glycuronate of potassium gives with phenyl hydrazin a crystalline combination with a melting point of 114° to 115° C. It reduces in the same manner with grape sugar, copper, bismuth salts in alkaline solution, and ammoniated silver solution. It decolorizes indigo solution. Equal molecules of glycuronic acid possess equal reducing power. Under the action of bromine there arises out of glycuronic acid, saccharine acid in the same manner as glyconic acid out of dextrose.

Urrhodinic Acid.—Robert Kirk[39] describes a new acid found by him in the urine of three brothers in one family, which gave the reaction of Boedeker's alkapton, assuming a dark color and greedily absorbing oxygen when alkalized, and which resembles in some of its reactions the glycuronic acid just described. There was frequent painless micturition, and the urine always reduced the copper salts with about five times the power of an equal quantity of glucose, but was without effect on the bismuth salts. His method of obtaining the acid was as follows: The urine was concentrated to one-tenth, or less, and then washed with several volumes of ether to remove the gummy and resinous bodies interfering with crystallization. Among substances so removed was a yellow body which appeared to be peculiar to this kind of urine. The ether and concentrated urine were next separated from each other, and the latter treated with dilute hydrochloric acid,—one-half per cent. of the anhydrous acid to the original amount of urine. The urine was again shaken up with ether when the new compound passed freely into it, communicating to it a yellow color, and from which it was afterwards deposited in a crystalline form. The crystals formed on the evaporation of the ether assumed the shape of large stellate groups, and sometimes also of sheaf-like bundles. In another and deeper watch glass they assumed the form of long thin prisms with oblique ends. They possessed a strong aromatic odor and brown color, although in mass they seemed almost black, with shining colorless points of needles scattering here and there. They were very soluble in water and ether, but rather more so in the former, and the solutions were of yellow color, becoming red when sufficiently concentrated. Kirk has been unable to identify the acid by any known to him, or of which he can find an account. It is regarded as being neither pyrocatechin nor protocatechnic acid. Kirk proposes to call it urrhodinic acid. The urine he investigated contained at least one-half per cent. of this acid.

Glycosuric Acid.—In this same category may be included the substance already alluded to as extracted by John Marshall,[40] and provisionally called by him glycosuric acid. Its reducing power is also greater than that of glucose, 0.6 c.c. of the undiluted urine being sufficient to reduce the cupric oxide in 10 c.c. of Fehling's solution, equivalent to 0.05 of glucose or expressed in glucose units, equivalent to 8.3 per cent. of glucose. Marshall says:—

"It is quite likely that this acid may occur more frequently in urine than is suspected, probably only in less quantity than contained in the urine just referred to, and to its presence possibly may be attributed the many peculiar and unsatisfactory reactions so often noticed when testing urine with Fehling's solution. Quite likely, too, that in some samples of urine the acid may be contained in sufficient quantity to produce a reduction with Fehling's solution in such a satisfactory manner as to be mistaken for glucose,—and thus many erroneous diagnoses of diabetes mellitus may have occurred.

"The urine from which the acid was obtained was of a brownish-red tint, perfectly clear and without sediment. [It responded to all of the copper tests, but not to the bismuth and fermentation tests, and had no influence upon polarized light when examined with the polariscope.] To isolate the acid the following method was employed:—

"The urine was treated with half its volume of plumbic tribasic acetate solution, and the resulting voluminous precipitate collected on a filter and washed several times with a mixture of equal parts of alcohol and water. The precipitate was then suspended in warm water and hydrogen sulphide passed through until all the lead was precipitated. After expelling the hydrogen sulphide from the filtrate by boiling, excess of plumbic carbonate was added, and the liquid was gently boiled several minutes, and then filtered while hot. The filtrate was concentrated on the water bath and then kept in a cool place to allow crystallization to occur. The crystals of the lead salt which separated were washed by decantation with a mixture of equal parts of alcohol and water and recrystallized from hot water. Finally, when sufficiently pure, they were dissolved in hot water and the lead precipitated by hydrogen sulphide, filtered, and the filtrate containing the free acid evaporated to dryness at about 70° C. The residue was extracted with ethyl ether, and the latter evaporated spontaneously. Several recrystallizations from ether, the final one from a mixture of ether and water, are necessary to obtain the acid in a fairly pure condition. The crystal mass was pressed between bibulous paper and again recrystallized from water. The acid thus obtained crystallizes in opaque white tetragonal prisms, melts at 140° C., and sublimes in same prismatic form, the crystals generally radiating from a centre. It is very soluble in water and ethyl ether, soluble in absolute alcohol and also in ordinary alcohol, sparingly soluble in chloroform, insoluble in benzol, toluol, and in petroleum ether. When its solution in ethyl ether is evaporated at a temperature of about 60° C., a slight claret-red tint is produced, which at times resolves into purple spots. This purple substance (somewhat resembling murexide) attaches itself to the crystalline mass, producing a beautiful appearance. The crystals, including the purple substance, dissolve in water, with a disappearance of the purple coloration. In the spontaneous evaporation of the aqueous solution of the acid no change of color is noticed.

"The acid does not contain sulphur or nitrogen, and is absorbed by animal charcoal. When the urine itself is passed through animal charcoal the filtrate becomes dark claret-red in color, and has lost its reducing property.

"Sodium hydrate gives a brownish coloration, beginning at the surface of the liquid (due to absorption of oxygen). Oxyphenic acid gives an almost similar reaction, only that a green coloration is first produced, which is not the case with the other acid. The brownish coloration noticed when the diluted urine containing the acid is added to Fehling's solution, is partly due to the action of the alkali of the Fehling's solution upon the acid. Picric acid causes no change. Upon the addition of sodium hydrate to the mixture of the acid and picric acid, a brownish coloration is produced similar to that produced by sodium hydrate alone. No reduction of the bismuth salt in Böttger's test occurs with the acid. The acid responds to Trommer's test, as also to Fehling's test. Argentic nitrate is reduced in the cold by the acid. The fermentation test fails completely. Its aqueous solution has no effect upon polarized light. Upon the addition in turn of a dilute neutral solution of ferric chloride, ammonium hydrate, and acetic acid, the play of colors from green to violet, and then to green as with oxyphenic acid, does not occur. It does not respond to the tests for hydrochinon. With a dilute neutral solution of ferric chloride a blue coloration is produced which very soon disappears. From this reaction the acid would appear to be a phenol derivative. It forms lead, barium, and calcium salts.

"The lead salt crystallizes in heavy needle-like prisms, melting at 209.5° C. It is soluble in hot water, insoluble in benzol, toluol, petroleum ether, absolute or ordinary alcohol, ethyl ether, and chloroform. It is decomposed when passed through animal charcoal, the acid remaining in the charcoal and the lead coming through with the filtrate as an insoluble white compound.

"On account of insufficiency of pure material—acid and lead salt—no ultimate analysis has thus far been made. In a short time I hope to have enough material for that purpose, and then a formula for the acid can be constructed, and more learned regarding its source in the human organism. However, two lead determinations in the lead salt have been made: 0.1466 gram air-dried lead salt gave 0.0717 gram $PbSO_4$, equivalent to 33.50 per cent. of lead. 0.1314 gram air-dried lead salt gave 0.0649 gram $PbSO_4$, equivalent to 33.66 per cent. of lead. Mean percentage of the two determinations, 33.58 per cent. lead, indicating that the acid has probably a high molecular weight.

"[The lead salt undoubtedly contains water of crystallization, but the amount has not yet been determined.]"

CREATININ.

Creatinin in Normal and Pathological Urine.—In opposition to Hoffmann, Grocco[41] finds that where muscular work is considerably increased the quantity of creatinin eliminated is increased. Even in the urine of babies in the first few months of life, and fed exclusively on milk, creatinin may be found in very small quantities. Fever in general leads to increased creatinin, but the relation between the degree of fever and the quantity of creatinin is not constant. In adynamic forms or with renal complications the creatinin diminishes. Alimentation also influences the quantity eliminated. In fevers with rapid wasting, the creatininuria is more marked. Hydræmia and cachexia lead to a diminution of creatinin, but in every case the cause and progress of anæmia must be taken into consideration, since with an equal degree of anæmia, the creatinin may be excreted in very different quantities and even in grave anæmia, there may be an increase due to the presence of fever. Jaundice does not lead, at least, necessarily, to a diminution of creatinin in the urine. In heart disease there is a diminution in the last stage of the cachexia; with commencing loss of compensation the creatinin may be increased. Diffuse parenchymatous nephritis and interstitial nephritis cause diminution of creatinin. In 3 cases of diabetes mellitus there was slight diminution also. In nervous diseases with muscular agitation creatinin increases. In others, with immobility, it tends to diminish. Amytrophic paralysis of the spinal and the peripheral region—poliomyelitis, atrophy and progressive muscular pseudo-hypertrophy, primitive multiple neuritis—give a general diminution of creatinin when the muscular atrophy is sufficiently extensive; but if the atrophy takes place rapidly the creatinin may be increased.

Phosphatic Diabetes.—Under this title, first applied by Teissier, of Lyons, Chas. Henry Ralfe[42] describes a condition in which there is a continuous and successive discharge of phosphates in the urine, attended with symptoms not unlike those of saccharine diabetes. Of the cases of this condition Teissier makes four groups: (1) It may be observed in certain functional derangements of the nervous system. (2) It may precede or accompany certain affections of the lungs. (3) It may exist with glycosuria or alternate with it. (4) It may run a distinct course by itself. Of 13 cases described by Ralfe, all except 2 occurred in male adults. The symptoms common to all, although varying in degree, were loss of flesh, aching rheumatic pains, chiefly affecting the lower part of the back and pelvic regions, a dry harsh skin, a tendency to boils, appetite generally ravenous, but in some cases, a morbid refusal of food. The important feature of these observations is that they go to show to be erroneous what has for many years been regarded, more particularly under the authority of Bence Jones, as almost an axiom, viz., that the phosphates passed in the urine never exceed those ingested with the food, and in fact are the accurate measure of what is ingested. Ralfe's well-earned reputation for accuracy must insure careful consideration of these observations, which are in confirmation of those of Teissier.

H. Anott,[43] of London, Canada, publishes some observations on the phosphates, in which he states that they are diminished in Bright's disease, in the late stage of chronic diabetes, where he suggests that they may have to do with diabetic coma; also in irritation of the nervous system, whether in the shape of disease, injury or overexcitement. He says the crystalline phosphate of lime is found mostly in chronic brain disease.

TOXICITY OF URINE.

Influence of Milk Diet upon the Toxicity of the Urine.—Charrin and Roger[44] made a study of this subject, experimenting with the urine of the dog and guinea-pig. The most important point of this study is that under the influence of a milk diet exclusively, the difference in the character of the urine disappears, and whatever the animal under experimentation, the dog, guinea-pig, or rabbit, the quantity per kilogram is the same,—the toxicity ranging at about 1.700 kilograms. This considerable diminution in the toxicity of the urine, under the influence of the milk diet, is in accord with the result of experience, accounting for the happy influence which the use of milk exerts in maladies where poisons play a part, and particularly in uræmia.

Toxicity of Non-febrile Pathological Urine.—V. Feltz[45] has recently presented a note to the Academy of Sciences in Paris on the toxicity of non-febrile pathological urine. Having shown that the properties of the toxic febrile urine are greater than those of non-febrile urine, and that these effects are not due to the density of febrile urine, he made a series of experiments on the effects of non-febrile urine from patients suffering with glycosuria, albuminuria, icterus, cancerous cachexia and anæmia. The urine was freshly filtered, analyzed and heated to the temperature of the dog's body before being injected. Intravenous injections were practiced, according to the method described by Fisher and Ritter in their experiments on uræmia. Feltz's experiments showed that glycosuric urine from non-cachectic patients is not more toxic than normal urine. Icterous urine from cases of organic diseases of the liver, albuminous urine from serious renal cases, and urine from patients suffering from cancerous cachexia or serious anæmia is considerably more toxic than normal urine. The symptoms observed, the kind of death which follows, and the conditions found at the post-mortem examinations, are similar to those observed when normal urine is injected in sufficient quantities to cause death. Feltz concludes that it is an increase in the noxious principles peculiar to normal urine, rather than the presence of new noxious elements claimed to exist in pathological urine, and that the salts of potassium contained in urine are the principal agents in uræmic intoxication.

GASES IN URINE.

Ralfe reports a case of this rare condition. A middle-aged man, for some weeks being treated for aortic disease in the out-patient department of the London Hospital, was finally admitted to the ward on account of weakness. His urine was examined on several occasions with negative results. Shortly after admission it was noticed that he was slightly delirious and was very drowsy; also that his urine, when freshly passed, had a specific gravity of 1024, and contained sugar. An hour later it was 1040, and when agitated, emitted bubbles of gas,—after which agitation the urinometer was forced up a couple of degrees. When the urine was poured from one vessel to another, the disengagement of gas was so great as to force the index of the urinometer quite above the fluid, even exposing the upper surface of the bulb. In a day or two the urine became free of sugar. Ralfe thinks it is possible that the excitement of leaving home may have caused an acute diabetes, while the drowsiness may have been a threatening of diabetic coma. Under such circumstances, the urine may have contained large quantities of acetone, which undergoing decomposition allowed large quantities of carbonic acid to escape. On the other hand he queries whether in a neurotic subject laboring under hysterical emotion, a large quantity of carbonic acid may not have diffused rapidly out of the blood into the urine, just as we know it does into the intestines in cases of phantom tumor, and in some cases of extreme flatulent distension. As the reaction of the urine was acid, the gas could not arise from the decomposition of urea. The gas was quite inodorous: hence it could not have been sulphuretted hydrogen.

Frederich Müller[47] reported a case of a serving woman, 29 years of age, with rapid phthisis, in whose urine sulphuretted hydrogen was present. The urine had a fæcal odor and at daily examination gave a decided reaction for sulphuretted hydrogen. There was a fetid vaginal discharge, at times diarrhœa, with stools containing sulphuretted hydrogen, and also a recto-vaginal fistula. Müller believes that in the majority of instances hydrothinura is a consequence of sulphuretted hydrogen fermentation in the urine. Its origin from the bowel and absorption of the kidneys, and from admixture of air and pus in the vicinity of the bladder is possible, but rare. Theodore Rosenheim,[48] as a result of recent experimental researches, concludes that bacteria are the causes of the decomposition of the sulphur-holding substance.

CHYLURIA.

Etiology.—Rossbach,[49] of Jena, has presented a valuable contribution upon a case of chyluria of indigenous origin. A young woman, aged 21 years, a factory hand, unmarried, fat and flabby, with compensated mitral insufficiency, and with marked hysterical symptoms, was the subject. Early in the course of the case the area of hepatic dullness was increased, and later became contracted. She complained of pains in the region of the liver, and also in the head and in the joints, which at times were swollen, œdematous and red. The fæces were solid and formed, with but a small amount of fat present, even during a prolonged fatty diet. Early in the case—and particularly at menstrual epochs—the amount of milky urine passed was markedly subnormal; later it rose to normal and on several occasions beyond this. Within a varying length of time after the urine was voided, clots of different, but always considerable, sizes would be found in it. These, however, did not, as in chyluria of tropical origin, become converted into a trembling gelatinous mass. The urine was always chylous, no matter when voided,—contrasting with that class of instances where the night urine is chylous and the day urine clear, or vice versa. Examination of the fluid showed at first from 23 to 32 grains of fat in the urine of 24 hours; but later in the case this quantity rose to as much as 150 or 160 grains. After ethereal extraction of the fat, the residue always presented two distinct solid portions, the larger of which was of a brownish color and melted at about 68° F. The other and smaller part always collected on top, was of a white opaque color and melted at about 145° F. In the ethereal extract were found fat, lecithine and cholesterine; albumin was also found, partly in solution, partly employed in emulsifying the fat. In his remarks Rossbach said that the condition of the patient, the absence from the urine of any structural elements of the urinary apparatus in a condition of fatty metamorphosis, would exclude such disease of this portion of the system; that the absence of sugar, the relatively small amount of albumin and large amount of fat (double the maximum amount in normal lymph), the absence of parasites from the blood and the urine, the small amount of urine voided, and the small amounts of sulphates and phosphates, would exclude the possibility of a lymph fistula. He spoke of the fact that finely emulsified fat when injected into the blood is excreted by the healthy kidneys, as tending to sustain his idea that in this case the fat was in the blood before it entered the nephritic system, and that the renal epithelium had separated it and retained the formed elements of the blood. Again, the absence of fat from the diet did not lessen the amount in the urine; the presence of lecithin as an intermediate step in the transformation of albumin into fat was significant; and the hepatic symptoms to the lecturer indicated a possible cause in a diseased condition of that organ.

Francotti[50] also reports a case of non-parasitic chyluria, occurring in a woman for the first time, in the middle of her second pregnancy, and reappearing in the third and fourth pregnancy. At this last time it remained for 18 months, clearing up under laxatives and rest in bed, and recurring upon exertion or prolonged standing. Then for four and a half years the urine was generally clear, becoming milky only on fatigue. Liver, spleen and heart were apparently normal. On abdominal examination a tumor of the size and shape of the kidney was discovered to the right of the median line. No parasites could be found in the blood or urine of the patient.

Yamane,[51] in Japan, a country in which chyluria is being found more or less frequently (Murata having been able to collect 82 cases since Baelz first noticed its presence in the empire in 1878), has found in a well near Nagasaki a parasite quite like the embryo of filaria sanguinis hominis.

HÆMOGLOBINURIA.

Etiology.—An important suggestion is made by Baker,[52] of North Carolina, who urges the probability of the existence of an additional factor in the so-called malarial hæmoglobinuria besides the malarial miasm itself. According to his observations this malady is marked frequently by a lack of periodicity in the temperature of the attack and in the attack itself, and by its appearance at times when and in localities where malaria is comparatively rare. He has found post-mortem in the intestinal connective tissue and capillaries, and in the liver, bacilli of a long slender shape, and regards their presence as necessary to the destruction of the red blood corpuscles, in the production of hæmoglobinuria. In an editorial upon Baker's researches the *North Carolina Medical Journal* offers an illustration in an endemic of malarial hæmoglobinuria in Southport, N.C., some years ago. The disease seemed to have little relation to the prevailing forms of malaria. It had not been known before and has not since reappeared. There was no increase in the intermittent or remittent fevers, but rather a partial abeyance. The seasonal changes favorable to malaria—warm dry spells alternating with moisture—did not prevail, nor were there such continuous winds as might have carried the poison from the neighboring bogs. More negroes were fatally ill than whites; the field hands in the rice plantations, among whom malaria is frequent, were not especially attacked; and finally quinine was not so effective as in case of the ordinary malarial fever.

Certain other writers[53] assert a relation between the vaso-motor nervous system and the malarial dyscrasia associated with hæmoglobinuria or hæmaturia, as well as with the other dyscrasiæ, as purpura or hæmophilia, on account of which there exists an impaired contractile power and elasticity of the blood-vessels under the *vis à tergo*. From such a therapeutic view Prof. Grancher, of Paris, asserts that the value of quinia is in its tonic influence upon the vaso-motor nervous system. Among this class of observers, Abercrombie, of London,[54] suggests a direct relationship between Raynoud's disease and paroxysmal hæmoglobinuria. In a certain number of cases reported, syphilis was credited by the authors as a predisposing cause, but no positive knowledge upon the subject has been presented.

No matter what the predisposing conditions may have been, a large proportion of cases are certainly directly referable to sudden chilling as the determining agent, especially when associated with forced muscular exertions. The individuals, according to Bamberger,[55] of Vienna, become feverish, are presently chilly, and sometimes shiver with the chill; then comes a feeling of heat and at times a perspiration follows. Frequently gastric disturbances are present, with headache; the urine is stained with the blood coloring matter. In a few hours, or at most a few days, the patient is recovered, and his urine returned to the normal; but a repetition of the exposure to cold causes a recurrence of the malady. In order to study the paroxysm it has become more or less customary to expose such patients, by plunging the feet into very cold water and then inducing violent muscular exercise. (Boas-Erlich method.)[56] Cases apparently depending upon the action of cold are reported by Bruzelius,[57] of Stockholm; by Lehzen,[58] from Leube's clinic in Würzburg; Demme,[59] of Vienna; and by Kobler and Obermeyer,[60] from Bamberger's clinic in Vienna. Of the seven cases comprised in these reports, three were known to have had syphilis. Bruzelius has seen four instances, the first occurring, in 1882, in a woman aged 35 years. She had had two previous attacks at intervals of four years, each time preceded by exposure to cold, to which she was quite sensitive. At the time of observation, even placing her hands on a cold surface exposed her to a recurrence of the malady. The second was in a man aged 32 years, syphilitic; it each time followed chilling from exposure to rain; the patient died eight years after first noted from a fatty degeneration of the heart. The third case occurred in a man of seventy-one years, who died two years later from pneumonia. The fourth case was a man of 34 years, syphilitic. He was able to induce attacks by immersing his hands in cold water for 20–25 minutes. Leube's case occurred in a girl of 26 years, syphilitic, who regularly had an attack if she worked out in the open air, and would sometimes be ill if she merely walked abroad. Demme's case was that of a boy, aged 5 years, who fell into a tank of cold water from which he was at once rescued. Two hours later he vomited matter streaked with blood, and an hour later passed brownish colored urine; five hours after his immersion he died in collapse. The case from Bamberger's clinic was a coachman, aged 49 years, with good family and personal history. Thirty years before the attack he was a soldier, compelled to perform long marches in heat and cold, but was not in the least affected. In November of '85 he first noticed that his urine was "black as coal," and at the same time he experienced a distinct chilly sensation. After being confined for a few weeks to a comfortable room, all traces of disease disappeared. During the carnival season he again sat upon his box, but no sooner had he resumed duty when the disease returned. He was comparatively well again during the spring and summer of '86, but after a severe wetting in the autumn, he was again disturbed. Experimental attacks were induced by plunging his feet into water at 50° F. for a few minutes, and then causing him to walk hastily in the open air. In none of these cases is excessive alcoholic indulgence recorded.

A case of paroxysmal hæmaturia was reported by Handford,[61] of London, due to bilharzia hæmatobia. When first seen, in November of '85, there were many ova and embryonal worms found in the urine. Again in March of '87 these were to be seen, but in much smaller numbers. An interesting discussion ensued in the Clinical Society as to how long the embroyos could exist in a single person unexposed to reinfection, or whether these parasites could multiply within the single host.

Baginsky,[62] of Berlin, has found a nematoid worm described under the name rhabdites, associated in causal relationship with a case of paroxysmal hæmoglobinuria in a boy 3½ years old. The urine and not the blood contained the worm. There were none found in the stools. The nematoid in question has a well developed œsophagus enclosed in a covering, in the dilated end of which chitin teeth are to be found.

Symptomatology and Pathology.—In the bulk of cases of paroxysmal hæmoglobinuria due to general non-miasmatic causes, the train of symptoms is tolerably constant.[63] There is a rise in temperature, first associated with chilliness, next with sensations of warmth. There is frequently sweating and nausea, sometimes vomiting. Within a few hours the urine becomes highly stained, and in most of the cases a slight jaundice occurs. In many instances a demonstrable increase of hepatic and splenic areas exists. In a certain proportion of instances, as in Bamberger's case, a series of nervous symptoms sometimes may be noted: the pupils become contracted and insensible to light, the sensibility of superficial areas becomes diminished, and the reflexes are altered. All these symptoms, under absolute rest for a few days at most, disappear entirely. The temperature during the attack rises as high as 104° F. or over, and the respiration and pulse are also disturbed. The patients complain of pains in the back and loins, aching of the limbs, and a feeling of lassitude. Considerable difference of opinion exists as to the origin of the jaundice that is more or less constantly present, certain observers regarding it as purely hæmatogenetic, and others as probably hepatogenetic. In close relation to this is the discussion as to the real nature of the urine, whether it is a primary hæmoglobinuria or secondary to a hæmaturia. Küssner[60] found by spectroscopic examination of the blood serum during the attack that there is a condition of hæmoglobinæmia present, and this is accepted by the opponents of the theory of the primary character of the affection. These latter, however, do not regard this hæmoglobinæmia as produced by a systemic cythæmolysis, but rather by reabsorption from hæmatic urine which is delayed in secretion. Experiments of Silbermann,[60] by injecting various substances into the blood, tend to support the idea of a primary destruction of red blood corpuscles, but there is no positive knowledge yet advanced. After direct injection of a solution of hæmoglobin into the circulation, Silbermann was able to produce a series of phenomena analogous to paroxysmal hæmoglobinuria, and from his observations decided that the free hæmoglobin acted upon the white corpuscles of the blood to the production of a fibrin-ferment; that this last coagulating the blood would produce widespread areas of thrombosis, or if the effect is less severe, of areas of slowing of the current. It is known from the experiments of A. Schmidt[64] that venous blood is richer in fibrin ferment than arterial, and we would therefore expect this pathological condition of thrombosis or slowing of the current to occur with greatest severity in the portal circulation, and this is borne out by Silbermann's observations during his experiments. Clinically, too, confirmatory results have been presented. Demme's case showed marked congestion of the liver and spleen upon post-mortem examination, and there were numerous ecchymotic patches in the submucous connective tissue of the intestinal tract. The tubules of the kidney were filled with detritus of broken down red corpuscles. Sanders,[65] of Münich, reports a case occurring in a child three days old from uncertain cause. It was healthy up to the morning of its fourth day, when it became jaundiced, the urine highly stained, and death following in a few hours. On post-mortem section the heart was normal, spleen swollen and dark, the interior occupied by a large hæmorrhagic infarct. The kidneys were injected, and dark with foci of broken down red corpuscles, the tubules full of pigment matter. Leube's case was constantly attended with increase in the area of liver and splenic dullness.

While from such observations it is not unlikely that at least a portion of the pathological process occurs in the venous system, it is probable also that other parts of the system may enter into the production of the same clinical appearances of the urine. Luca,[66] in a series of experiments upon hæmatogenetic jaundice, concludes that jaundice sometimes occurring in the treatment of the affections by blood transfusion is not a purely hæmatogenetic one, but due in part to mechanical effects of swelling of the liver. The condition of the kidneys in the above cases (and in cases reported by Baker) on section indicate a possibility of their being a seat of change. Lewin and Rosner,[67] of Berlin, in investigating the spectra of bloody urine, have found that the absorption bands and spectrum lines of the blood products in such cases vary grealy and are of the opinion that very marked changes from the usual spectroscopic appearance of hæmatic urine may occur without the existence of a primary cythæmolysis which the theory of a purely renal hæmoglobinuria supposes.

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PSYCHOLOGICAL DISEASES.

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BUT little material progress can be recorded in the domain of psychological medicine. The administrative aspects of lunacy, which are of but slight value to the medical reader, and of no lasting scientific worth, continue to take up a large portion of the journals devoted to the subject. Undoubtedly the most interesting developments of the year relate to the phenomena of functionally perverted mental life observed in connection with hysteria, mental suggestion and hypnotism.

The strange and novel facts mainly observed by French investigators have not yet been sufficiently well systematized to admit of a philosophical explanation. The individual signs and features are for the present merely being catalogued. Some of them are of a most startling character. Thus, Babinski[1] claims to have established as a fact that certain manifestations of hysteria may be transferred from one person to another, under the influence of a magnet, even where the two subjects are separated by a considerable distance and mutually ignorant of each other's presence. In this way hemianæsthesia, hemiplegic monoplegias, paraplegia, coxalgia and mutism can be artificially inculcated in a person previously free from these symptoms, while they disappear in the person who previously had them. The two subjects in this experiment bear the same relation to each other, that the opposite halves of the body of one and the same person do in the case of ordinary physiological or pathological *transfer*. Babinski has gone one step further into this remarkable realm of inquiry. He claims to have shown that even the signs of organic nervous disease may be similarly transferred to a subject free from such disease, but previously hypnotized. The transfer is in this instance, however, not as complete as in the previous one, and the symptoms of the sufferer from the organic affection are not thereby modified.

Singular as these facts seem, what shall be said of the newer observations relative to hypnotization at a distance? Pierre Janet[2] and Charles Richer[3] claim that after employing every possible safeguard against sources of error, a certain subject named Léonie could be hypnotized by a person at a great distance, notwithstanding that she was unaware of the experimenter's existence or the place where he was. She became somnambulic within from five to seven minutes after Richer commenced to exert his influence.

Luys[4] has even claimed that medicinal substances could act in the same mysterious and impalpable manner, and a commission appointed by the Academy of Medicine to test his proposition has surprised the scientific world by resigning its trust. Voisin[5] has followed up the experiments of Bourru and Burot,[6] who confirm Luys on the same subject they experimented with and found that their claim and that of Luys were altogether fallacious and that the results supposed to have been obtained could all be accounted for as the consequence of suggestion, and auto-suggestion. This seems to us to be the best disposition to be made of a question which was fittingly inaugurated in the same centre of civilization which honored Mesmer and Cagliostro a century ago.

The wonderful effect which can be excited through mental suggestion in hypnotized subjects had early suggested its employment in the treatment of mental disorders. Braid, over forty years ago, claimed to have thus cured a case of what he terms "monomania." Within the year rose-colored reports of its efficacy have come from the Parisian asylums. Many intelligent critics hesitate to accept the results obtained by Charcot and his pupils, on the ground that so many trained subjects have been produced in Paris and its vicinity as to materially decrease the value of the fruits obtained from a soil thus artificially cultivated. Forel,[7] one of the ablest psychiatrical thinkers of the day, places the methods employed by Liebeault and Bernhard,[8] of Nancy, far above those of the sensational school of Paris, and this after careful personal observation. The investigators cited appear to be able to hypnotize from eighty to ninety per cent. of normal mankind! They find that by "suggesting" one and the same dream sufficiently often to a hypnotized person, it finally exerts an effect on the subject's mentality, and even on physical states, which extends into the waking state. Thus they succeeded in relieving muscular paralyses, anæsthesias and retention of urine due to functional disorder. It is even claimed (but Forel does not vouch for this fact from personal observation) that they have made deaf-mutes to hear! Forel, following the precedent set by Braid, Voisin and Flechsig, attempted to utilize this influence for the benefit of the insane inmates of the Swiss asylum under his charge. He found that the insane resist the hypnotic influence more than the sane do, but concludes that the new therapeutic measure which can be carried into effect by this means, is of greater importance than sceptics, who are mostly recruited from the ranks of the Germans, believe. A brief summary of his results as actually obtained would seem to justify the skepticism he attacks. The most favorable of these were (1) the relief of menstrual pain and production of sleep in one subject; (2) the breaking up of the alcoholic habit in a chronic drunkard, who later joined a temperance society; (3) an amelioration of the hysterical character; (4) disappearance of hallucinations during an induced somnambulism in a maniacal paranoiac: "unfortunately" they reappeared in undiminished intensity. The maniacal spells, however, could always be inhibited by ocular fixation and an imperative command accompanied by a stroke, from which combined procedure a deep sleep resulted; (5) nostalgia was relieved in one case. We believe that the above fairly represents what can be honestly claimed for hypnotism as a therapeutic measure in insanity. It cannot be denied that in a recent case, the repeated induction of dreams conflicting with a growing delusion might favorably modify the latter. But to attribute the taking of temperance pledges, modifications of the hysterical character and disappearance of homesickness to its influence, when all of these are spontaneous daily occurrences in and out of asylums, seems to have been done without duly weighing the question of coincidence. A supplement to Forel's article admits that in one case where hypnotism appeared to exercise a good effect, the latter wore off, subsequently to sending his article to press. This new method has now been before the alienist fraternity a year, but we can not find any records of well-observed cases, where its effect was radical and patent, and where other co-operative factors could be excluded. Such a case as the following, reported by Krafft-Ebing without any doctrinal purpose, seems to us to furnish an *experimentum crucis:* A female paranoiac who suffered from a distressing form of sexual perversion (abhorrence of persons of the opposite sex), and in whom the hypnotic state could be induced by staring at her, having her fixate a pencil, pressing her forehead or producing a monotonous noise, was offered the hand of princes, and other desirable partners in marriage, while hypnotized. She repulsed these offers as firmly as she resisted the suasions of males in the waking state, declaring that she never would marry a man.

The subject of the causation and pathology of paretic dementia continues to receive careful consideration at the hands of European alienists. The attention of etiologists has been closely attracted to the part played by syphilis. The mass of authorities is in favor of regarding it as one of the most important features. Dietz[9] concludes that when found in subjects under the thirtieth year, syphilis is almost always an etiological feature. Rieger proves by figures that persons infected with syphilis are over sixteen times as liable as non-syphilitic patients to develop paretic dementia. Dietz also contributes a valuable table, showing how late after the primary affection the disease breaks out. Of thirty-nine cases, twenty developed paretic dementia between one and ten years after contracting syphilis; seventeen between ten and twenty, and two later than twenty years. A remarkable feature in a number of cases cited by both observers, as well as by Eickholt and Siemens,[10] is the failure of anti-syphilitic treatment,—either unconditional or after a previous deceptive success in the shape of a remission. In one case a complete recovery, enduring nine months, was obtained under specific treatment; but at the end of that time a series of almost uninterrupted epileptic convulsions terminated life. It would appear from the records made by German physicians, as also from the careful study of syphilitic psychoses, made by Savage,[11] that the enthusiastic views of Fournier as to the curability of luetic paresis are not justified by facts.

It had become regarded as a canon of psychiatry that paretic dementia shows a tendency (increasing with each decade) to attack young persons; in other words, that it extends its ravages in urban communities by the early operation of overstrained and other debilitating as well as toxic causes. The enormous clinical material at the Berlin General Hospital has been thoroughly sifted by Thomsen,[12] with special reference to this point, and he finds that his figures, which cover the years between 1874 and 1886, do not support the assumption stated. The paretics admitted in the latter year were of the same age as those admitted in the former. A similar study of the insane population at the Charenton Asylum, during a nearly corresponding period, opposes another oft-repeated statement as to the rapid increase of this disease. The admissions of paretics in 1879 and 1886 were nearly the same, and bore the same relative proportion to the total admissions. Still Christian,[13] who furnishes these figures, is inclined to believe that when our reliable statistics shall have covered more than two decades, a slight increase may become apparent. He believes that of the predisposing causes, the moral ones are most important, finds the disease to be more frequent among the married than the unmarried, in the military than in the civil population, and that heredity plays a greater rôle than is generally supposed. In conflict with the German authorities, he regards the influences of alcoholic, sexual and tobacco excesses, as well as of syphilis, to be very doubtful. The general result of the studies made in this field during the year is to the effect that paretic dementia is not due to single causes, but to a combination of several: that any injury, toxic, traumatic or nutritive, or an inherited instability, may act as a predisposing cause, whose sinister effect is precipitated by the three W's, namely, Wine, Women and Worry, either singly or in combination, and that excesses in tobacco favor their operation.

The morbid anatomy of this disease has been studied chiefly in its minute features. Notorious atrophy of cortical nerve elements is graphically represented by Kronthal.[14] Palmer[15] figures what he designates as protoplasmic exudations in acute cases. Unless his excellent illustrations be susceptible of misinterpretation, it would seem as if what he thus describes were the well-known spider-cells of Lubimoff, and others. These may furnish hollow prolongations communicating with the lumina of cortical capillaries, and thus lead to the formation of numerous vascular channels, which, as Kronthal again points out, are so prominent in sections of the paretic cortex. With regard to the origin of these spider-cells, it may not be uninteresting to note that Forel[16] (working with the improved methods of Golgi) inclines to believe that *some* at least among them are not newly-formed elements, but small shrunken nerve cells.

The discovery by Tuczek of a noteworthy diminution of the nerve fibres contained within the cortical gray matter is now followed by a critical review of this question at the hands of Zacher,[17] who, on the whole, confirms his predecessor with more perfect staining methods. He demonstrates that the wasting of these nerve fibres is a slow primary parenchymatous degeneration. At first the fibres become very narrow, and those which retain their normal diameter do not stain well. Numerous varicosities develop, and the myelin becomes crowded together in masses, leaving intermediate sections of the axis cylinders bare. Soon the latter appear brittle, or irregularly contorted in course, and occasionally corroded in their contours. There is no strict uniformity in the progress and distribution of this change. Usually the surface layer of the cortex shows the most advanced degeneration, but often the latter is simultaneously established in the deeper strata. The frontal lobe appears to be the region most early involved, but, differing from Tuczek, Zacher declares that the parietal zone may be affected nearly at the same time, and in several cases found the changes to be irregularly or diffusely scattered over the cortex in general. The former writer stated that the intensity of the morbid process is in direct proportion to the duration of the case, and the degree of dementia. Zacher on the contrary finds that it may be both extensive and far advanced in a case of ten months' standing, as compared with one which had lasted seven years. Nor is it altogether limited to paretic dementia. It is found (less marked, perhaps, in intensity) in senile and alcoholic subjects. The latter occurrence induced Zacher to venture a surmise to the effect that the intra-cortical atrophy of nerve fibres may (like the corresponding change found in the peripheral nerves in alcoholism) be due to the direct effect of that nerve poison on nerve tissue. The conclusion drawn from these observations is that it would be premature to venture a definition of paretic dementia as an invariable pathological entity. Two different changes which may progress independently of each other are noted: the one is the essential atrophy of the fibres just mentioned, and is not related to the meningeal or vascular changes; the other consists in disintegration of nerve cells. Formation of shining granules and detritus appears to predominate in those cases where atheromatous degeneration of the blood-vessels is present. The former seems to play a decided rôle in the general atrophy made manifest in the diminished brain weight of paretics; for this is lowest where the fibre atrophy is most marked, and highest where it is not marked, or where inflammatory changes predominate.

A more extensive change in the associating fibre tracts, and of the same histological character is described by Friedmann:[18] it is confirmatory of the atrophy of sub-cortical associating fasciculi described ten years ago by the reviewer. Friedmann has made his investigations on specimens hardened in Miller's fluid, and may thus have failed to avoid certain sources of error involved in this method. Even in health, different color shades are produced in the white substance by this reagent. Little progress has been made in the study of the spinal changes of paretic dementia. Furstner[19] sums up the results obtained in this field by Westphal, Zacher, Schultze and himself, and discriminates between the following types of tract degeneration: (1) The ordinary type in which a lesion of the posterior column predominates and tabic symptoms develop. (2) The spastic type, where there is degeneration of the crossed pyramid tract in advance of the degeneration in the posterior columns. In one case, however, where there were spastic contractures involving all four extremities, no corresponding lesion was found. (3) In a few cases the direct cerebellar tract was found to be degenerated in company with the cross-pyramid tract, while the posterior columns were quite healthy. A marked feature of these cases was a rapid emaciation of the patients. This would seem to sustain those who regard the function of this tract as vegetative. With regard to the atrophy of the cross-pyramid tract, Furstner agrees with the mass of authorities in regarding it as disconnected from the cerebral lesion; but Forel describes a case (paralleled by one of the reviewer's) in which there was a direct relationship between the most atrophic cerebral hemisphere and the crossed will tract.

Very little has been added to our knowledge of the exact symptoms or physical signs of insanity. The more important ones studied are the changes of the pupil. Mocli,[20] Thomsen and Siemerling[21] have examined thousands of patients at the Berlin Charité. They find that in paretic dementia, the majority of the patients present a pupil of medium width: that variation in the direction of mydriasis is more rare than abnormal myosis. In

7 per cent. of the cases the pupil is found to be less than two millimetres wide. In these cases the outline is usually angular. A difference between the two pupils of opposite sides is common, but does not usually exceed one millimetre. Sometimes unilateral mydriasis occurs, but myosis is scarcely ever unilateral. It was found that taking the insane as a whole, the Argyll-Robertson pupil was found in but $1\frac{4}{10}$ of 1 per cent. These patients were paretics, or on the way to the development of paretic dementia. Of 500 paretics, 47 per cent. had lost the light reaction of the pupil; in 4 per cent. it was doubtful, and in 10 per cent. minimal. Among insane women the Argyll-Robertson pupil was found in 60 per cent. of the paretics, and in $1\frac{8}{10}$ of 1 per cent. in the non-paretics; and both Thomsen and Siemerling agree in stating that of the small number of non-paretic insane patients presenting the Argyll-Robertson pupil, the greater number appertained to traumatic forms, to syphilitic insanity and to senile dementia. It occurred in a very small per cent. of paranoiacs, in 2 per cent. of the alcoholic cases, and temporarily in a few cases of post-epileptic stupor.

Praiseworthy attempts to obtain characteristic representations of special types of insanity have been made by American alienists, notably by Noyes.[22] It is a question, however, whether a disorder like insanity, which (as far as the facial expression is concerned) involves not alone the specific alterations produced by disease, but also the modifications which the individual *facies* of the patient undergoes, can be advantageously represented by this method. From several of these examples lying before us we are inclined to believe that great care will have to be exercised in selecting cases. Members of the same race only should be utilized for a single photograph when attempting to derive the typical disease expression. Thus, if 25 paretic dements be photographed, and 23 of these be Celts or Saxo-Americans, while 2 are French, or Israelites, the strongly marked features of the latter will preponderate and overwhelm and obliterate the finer features of the disease shown by the majority. Avoiding this source of error, it is possible that this new method may have a future.

The chief contributions to morbid anatomy of insanity have been recapitulated under the caption of Paretic Dementia. The only other of importance relates to Idiocy.

Koch,[23] of Copenhagen, has made a minute study of a case of idiocy resulting from the use of the forceps in delivery. He found a cicatrix of the left parietal tuberosity, corresponding to the healed fracture of the bone, two centimetres in length. The dura underneath was normal; the pia showed numerous granulations, particularly toward the median line, where it had become adherent to the cortex. The left hemisphere was considerably the smaller, and on passing over either hemisphere, firm, small, nugget-like bodies could be felt; the largest were as large as cherry pits. They were limited to the gray substance, and showed a greater resistance to the knife. On closer examination they were found to be due to the hypertrophy of the neuroglia, which was particularly noticeable in the outermost layer of the cortex. In these patches, irregularly contorted fibres were found, round cells and increased nuclei. The author compares it to the multiple tuberous sclerosis of Bourneville and Bruckner.

A careful summary of our clinical knowledge of those strange mental phenomena found both in the asylum and the non-asylum population, known as imperative conceptions, has been made by Freidenreich.[24] He classifies these symptoms under the following heads: (1) Cases corresponding to the typical *maladie du doute* of Falret. (2) Cases corresponding to the Grübelsucht of Griesinger. (3) Cases occurring spasmodically like a tic. (4) Cases corresponding to the *délire émotif* of Morel. (5) Cases where the imperative conceptions constitute a veritable insanity. In all cases there is an associated irritability of the nervous system, and indeed the chief features, that is, the imperative conceptions and fears, are common signs of nervous weakness manifested in other directions than in those which are specifically recognized as cases of mental disorder. Agrophobia, claustrophobia and astrophobia constitute, as it were, transitions from the ordinary vague fears of nervous people to those more intense imperative conceptions in which the patients are seized with spells of terror. It is not always (as has been claimed by most authors) that the imperative conceptions are foreign ingredients of the patient's mentality, and disconnected from his other thoughts. They may attain an actual dominion to such an extent that the patients fail to recognize their fallacy, and in other cases they impair conceptional vigor. As soon as the will power is involved, Friedenreich regards the patient as having entered the domain of lunacy proper. As regards the causation he finds that heredity is the most important predisposing factor; that it is more common in youth than in old age; that it is of equal occurrence in both sexes, and more frequent in the educated than in the uneducated. The prognosis he finds to be bad as to recovery, good as to preservation of intelligence, and that there is really only one great danger, and that limited to a certain class of cases,—namely, suicide.

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